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A COMPARISON OF EXERCISE RESPONSES OF MALES AND FEMALES DURING ACUTE EXPOSURE TO HYPOBARIA

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DURING ACUTE EXPOSURE TO HYPOBARIA

BY
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DISSERTATION

Submitted in Partial Fulfillment of the
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in the Graduate School of
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Philip Richard Elliott, Ph.D.
Department of Health, Physical Education and Recreation
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This study was designed to investigate the effects of acute altitude exposure on selected pulmonary, cardiovascular, and metabolic variables in men and women during submax and max work. Seventeen male and 20 female physical education students were randomly selected for inclusion in the investigation which took place over an 11-wk period. All testing was conducted in a hypobaric chamber with each subject being tested initially at the terrestrial altitude of 1576 m to which they were acclimated. Subsequent tests were conducted at simulated altitudes of 2743 m and 3962 m.

The subjects pedalled a bicycle ergometer at a rate of 60 rpm and a power load of 90 kpm/min for the first 3 min after which the load was augmented by 90 kpm/min at the end of each minute until pedalling rate could no longer be maintained. When this point was reached, the test was terminated and a 10-min recovery period commenced. Heart rate, blood pressure, and breathing frequency were recorded the last 20 sec of each minute

and 30-sec samples of expired air were collected periodically until exercise was terminated. During the recovery period, heart rate was recorded during submax and max exercise as well as recovery. Minute ventilation, breathing frequency, and oxygen pulse were calculated for submax and max exercise. Mean arterial blood pressure was recorded for submax exercise only. Oxygen consumption, respiratory exchange ratio, and ventilation equivalent for oxygen were calculated for max exercise only. Analysis of covariance with repeated measures was employed to test for differences between the sexes during submax and max exercise at the two simulated altitudes. Ventilation (BTPS) was the only variable during submax work to exhibit a consistent difference between males and females in that women had a lower ventilation than men at both 2743 m and 3962 m. The greater ability of women to protect the acid-base balance at altitude was advanced as a possible explanation for this difference. From 1576 m to 2743 m, the percentage changes in submax heart rate and oxygen pulse were found to be different between the sexes. A theory for this apparent advantage of women as compared to men to tolerate minor hypoxic stress was advanced. It was suggested that women, at lower elevations, have latent reserve in local muscle perfusion which is mobilized when hypoxia reaches sufficient levels for massive vasodilation.

During max work, oxygen consumption was affected more by altitude in women than in men. This was explained by their inability to increase ventilation. Max ventilation which remained lower in women was attributed to disparities in physiological limits, acid-base balance, oxygen cost of ventilation, and aerobic capacities between the men and women in this study. The variance in aerobic capacity as well as in airway resistance was also thought to be responsible for the aforementioned differences in oxygen [illegible]mption and the altitude-sex interaction which was [illegible] for this variable. The difference in oxygen pul[illegible] a manifestation of the observed changes in max oxygen pulse.

It was concluded that, for the subjects in this study, females tolerated hypoxia better during submax work than did men. During maximal work, the men apparently had a better tolerance for hypoxia than did the women.

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DEFINITION OF SYMBOLS

$AVDO_2$	= arterio-venous oxygen difference
BTPS	= body temperature pressure saturated
BP	= blood pressure
$\overline{BP}$	= mean blood pressure
CaO_2	= oxygen content of arterial blood
f	= respiratory frequency
FEV_1	= 1-sec forced expiratory volume
Hb	= hemoglobin
Hct	= hematocrit
HR	= heart rate
LA	= lactic acid
l/min	= liters per minute
MBC	= maximum breathing capacity
OP	= oxygen pulse
$PaCO_2$	= arterial carbon dioxide pressure
PAO_2	= alveolar pressure of oxygen
PaO_2	= arterial oxygen pressure
P_B	= barometric pressure
PO_2	= partial pressure of oxygen
RER	= respiratory exchange ratio
SL	= sea level
STPD	= standard temperature and pressure dry

SV	=	stroke volume
$\dot{V}$	=	minute ventilation
$\dot{V}_A$	=	alveolar minute ventilation
VDS	=	dead space ventilation
VT	=	tidal volume
$\dot{V}/\dot{V}O_2$	=	minute ventilation equivalent for oxygen
$\dot{V}O_2$	=	minute oxygen consumption
wks, mos, yrs	=	weeks, months, years

CHAPTER I

THE PROBLEM

Introduction

In July, 1976, female cadets began approximately 6 wks of intensive basic military training and physical conditioning at the United States Military, Naval, and Air Force Academies. This marked the first time in the history of these schools that they have been open to female students. The physical training is extremely demanding on entering cadets and the women are expected to perform on a level which is as demanding as that experienced by the male cadets.

A unique problem for the female cadets at the Air Force Academy was caused by the reduced barometric pressure due to the school's elevation of 2196 m. At this altitude, the dry ambient partial pressure of O_2 (PO_2) drops to 122 mm Hg from its sea level (SL) value of 159 mm Hg. The alveolar partial pressure of O_2 (P_AO_2) is similarly reduced from 103 mm Hg to approximately 72 mm Hg. Thus, in blood with normal hemoglobin concentration, the O_2 content of arterial blood (CaO_2) is reduced from approximately 20 volume percent to about 18.5 volume percent.

This means that the greatest possible arterio-venous O_2 difference ($AVDO_2$) could be decreased by some 7% (62).

It is well established that such a reduction in PO_2 is accompanied by a decrease in max work performance when the effort is mainly aerobic (67). Buskirk (13) has estimated a decrease in max O_2 consumption (max $\dot{V}O_2$) of 3.2% for every 1000 ft (305 m) increase in altitude beyond 5000 ft (1524 m). This would translate into a decrease of approximately 7% at the altitude of the Air Force Academy which is in agreement with Shephard's estimate for the comparable altitude of Mexico City (62). Consolazio (17) reported decrements in max $\dot{V}O_2$ of 7% and 17%, respectively, at 5280 ft and 11400 ft (1609 m and 3475 m). Interpolation indicates a decrease of approximately 10% at an elevation of 2195 m. Similar reductions have been reported by others (43,44).

The question can legitimately be raised as to whether women exercising at altitude will show performance decrements which are qualitatively and quantitatively similar to those observed in men. Although different physiological responses to reduced barometric pressure have been reported for men and women (20,24,27,33,37,35, 36,63), only three studies (20,21,56) involved exercise at altitude and each had drawbacks which precluded a definite answer to the question of sex differences.

One study (56) utilized but one female subject and the findings were not differentiated by sex. Another investigation (21) included two women of a total of five subjects, but ages and physical condition varied considerably in that both females were 29 yrs old, while the males ranged in age from 20 yrs to 75 yrs. In addition, the youngest male had experienced complications following surgery and, therefore, taken part in only light physical activity for 8 wks prior to the first exercise test. The third investigation (20) was conducted at high altitude and compared 10 females and 14 males who were native residents of the high Andes with 7 female and 13 male newcomers from lower elevations. The exercise information was of limited value, as it consisted of only 5 min of submax work, the level of which had been predetermined for each sex rather than related to individual capacities.

Adequate determinations of change in the work capacity and cardiovascular and pulmonary function of women during exercise at moderate altitudes require the fulfillment of certain requisites. The sample must be large enough to detect significant differences if they exist, the exercise intensity should be relative to capacity, and exercise duration should be long enough to clearly elicit changes in the physiological functions of interest.

Purpose

This study was conducted for the purpose of investigating possible differences between men and women in work performance decrements as well as changes in cardiovascular and pulmonary functions while exercising during exposure to reduced barometric pressures. The effects of simulated altitude on heart rate (HR), blood pressure (BP), oxygen consumption ($\dot{V}O_2$), ventilation ($\dot{V}$), and other pertinent cardiovascular, pulmonary, and metabolic variables were explored during submax and max exercise in the hypobaric chamber of the Human Performance Laboratory at The University of New Mexico.

Hypotheses

The hypotheses which were formulated for this study pertain to expected sex differences, or absences thereof, in physiological adjustments during submax exercise, max exercise, and the recovery following max exercise at moderate altitude. All hypotheses have been stated as null due to the use of inferential statistics. However, based on a review of related literature, it was expected that some of the hypotheses would be rejected. Specifically, the hypotheses referred to quantitative sex differences in the alterations of certain variables which could be expected as a result of acute exposure to moderate altitudes (2743 m and 3962 m) as compared to low altitude (1524 m). The hypotheses have been stated in such a manner that rejection will occur if a significant difference is found in any of the following: (1) main effects of sex, (2) interaction of sex and altitudes, and (3) simple effects of sex at either altitude. For statistical significance, a probability of less than 0.05 was chosen. Hypotheses I-V refer to standardized submax exercise during acute exposure to moderate altitudes.

Hypothesis I

There is no difference between males and females with regard to altitude effect on submax heart rate.

The Fick equation, $HR = \frac{\dot{V}O_2}{SV \times AVDO_2}$, illustrates the dependence of HR on certain cardiovascular and metabolic variables. A higher submax HR will be expected in both sexes while exercising at a decreased ambient PO_2 (1,9,17,62,65). This effect would likely be magnified in women if their ventilatory increase is indeed a result of a higher respiratory frequency (f) and a much smaller increase in tidal volume (VT) than is the case in men (20). This would result in a larger dead space ventilation (VDS) in women and, consequently, a smaller fraction of their inspired air being available for gas exchange in the alveoli.

Submax $\dot{V}O_2$ and stroke volume (SV) during acute exposure to moderate altitude have not been adequately studied in women. This investigator failed to see any reason why these variables would be affected differently in women, as compared to men. Although SV may or may not be affected by altitude (29,74), there are no indications that sex differences would exist. However, the decrease in alveolar ventilation ($\dot{V}_A$) in women upon ascension to higher altitudes (20) would result in smaller P_AO_2 and arterial O_2 tensions (P_aO_2). This in turn would adversely affect the arterial O_2 saturation (20) and, as a result, the $AVDO_2$. Due to the shape of the O_2 dissociation curve, this effect would be drastically amplified at altitudes above 3000 m. Therefore, since women would suffer a greater drop in C_aO_2 than men and since an increased O_2 transport to the mitochondria is probably a matter of acclimatization over a period of time (48), the greater decrement in $AVDO_2$ could be compensated for by means of a greater increase in HR. Therefore, an increase in HR was expected in both sexes with the females showing the greater increase. Hence, rejection of the null hypothesis was anticipated.

Hypothesis II

There is no difference between males and females with regard to altitude effects on submax mean blood pressure.

Increased arterial pressures have been documented in both sexes at rest (37) and similar adjustments could be expected during exercise at a decreased ambient PO_2. According to the rationale for Hypothesis I, women would experience a greater drop in P_aO_2 and a greater increase in the arterial CO_2 pressure (P_aCO_2) which would result in vasodilation. Therefore, an increase in $\overline{BP}$ was expected in both sexes with the increase being smaller in women. Thus, rejection of the null hypothesis was anticipated.

Hypothesis III

There is no difference between males and females with regard to altitude effects on submax breathing frequency.

Cudkowicz et al. (20) found a decrease in f during submax exercise in both sexes at altitude. Their data showed this effect to be magnified in women. Therefore, a decrease in f was expected in both sexes with the decrease being greatest in the females. Thus, rejection of the null hypothesis was anticipated.

Hypothesis IV

There is no difference between males and females with regard to altitude effect on submax minute ventilation BTPS ($\dot{V}_{BTPS}$).

An increase in $\dot{V}$ would be expected in both sexes while exercising at a decreased ambient PO_2. Reduced ambient PO_2 would necessitate a larger $\dot{V}$ to compensate for the decreased number of O_2 molecules per volume of air. The greater increase in VDS observed in women during submax work (20) would indicate a greater need for increased $\dot{V}$. Therefore, an increase in $\dot{V}$ was expected in both sexes with the increase being greatest in the females. Thus, rejection of the null hypothesis was anticipated.

Hypothesis V

There is no difference between males and females in regard to altitude effect on submax oxygen pulse (OP).

From the rationale provided for Hypothesis I, it follows that the OP may be expected to decrease in both sexes, but to a greater extent in women. Thus, rejection of the null hypothesis was anticipated.

Hypotheses VI-XIII refer to standardized max exercise during acute exposure to moderate altitude.

Hypothesis VI

There is no difference between males and females in regard to altitude effect on max oxygen consumption ($\dot{V}O_2$).

A decrease of 3.2% in max $\dot{V}O_2$ for every 1000 ft (305 m) increase in elevation above 5000 ft (1524 m) has been reported in men (14). The expectation of a greater decrease in women is supported by the previous discussion on $AVDO_2$ (Hypothesis I). In addition, as discussed in the rationale for Hypothesis XIII, the decreased buffering capacity of women (33), as compared to men, would result in a more reduced exercise time and, consequently, a greater reduction in max $\dot{V}O_2$. Therefore, a decrease in $\dot{V}O_2$ was expected in both sexes with the decrease being greatest in females. Thus, rejection of the null hypothesis was anticipated.

Hypothesis VII

There is no difference between males and females in regard to altitude effect on max heart rate (HR).

Reductions in max HR over SL values are usually not found at elevations less than 4000 m (28,30). Since max HR, by definition, cannot be accelerated further and since the vagal tone has been reported to be decreased during max work at altitude (59), there is no reason to anticipate a different reaction in women. Therefore, males and females were expected to exhibit the same max HR at altitude as they had at 1576 m. Thus, acceptance of the null hypothesis was anticipated.

<u>Hypothesis VIII</u>

There is no difference between males and females in regard to altitude effect on max breathing frequency (f).

Expectations of decreased f during max exercise may be supported for both sexes by the submax findings of Cudkowicz <u>et al</u>. (20). Hypotheses I and VI indicate that women were functioning closer to their ventilation maximum during submax exercise and, consequently, would be expected to sustain max exercise for a shorter period of time. This has been used to explain findings of smaller increases in max breathing capacity (MBC) of women as compared to men (63). Therefore, a decrease in f was expected in both sexes with the decrease being greatest in females. Thus, rejection of the null hypothesis was anticipated.

<u>Hypothesis IX</u>

There is no difference between males and females with regard to altitude effect on max minute ventilation ($\dot{V}$).

An increase in $\dot{V}$ was expected in both sexes while exercising at a decreased ambient PO_2 with a greater decrease anticipated in females. The rationale for Hypothesis VIII is applicable. Thus, rejection of the null hypothesis was expected.

Hypothesis X

There is no difference between males and females with regard to altitude effect on max respiratory exchange ratio (RER).

This hypothesis was offered in accordance with Hypotheses VI and IX. In addition, a traditional criteria for work to be maximal is an RER in excess of 1.0, and there is no indication in the literature of sex differences in this respect. Therefore, males and females were expected to exhibit the same max RER at altitude as they had at 1576 m. Thus, acceptance of the null hypothesis was anticipated.

Hypothesis XI

There is no difference between males and females with regard to altitude effect on max oxygen pulse (OP).

A reduction in OP was expected in both sexes while exercising at a reduced ambient PO_2. The rationale for Hypotheses VI and VII is applicable. Therefore, a decrease in OP was expected in both sexes with the decrease being greatest in females. Thus, rejection of the null hypothesis was anticipated.

Hypothesis XII

There is no difference between males and females in regard to altitude effect on max minute ventilation equivalent for O_2 ($\dot{V}/\dot{V}O_2$).

The rationales for Hypotheses VI and IX are applicable. The increase in max $\dot{V}$ is expected to be greater than the decrease in $\dot{V}O_2$ for both sexes. Thus, $\dot{V}/\dot{V}O_2$ is anticipated to increase in both males and females as barometric pressure decreases. This change was expected to be similar for both sexes resulting in acceptance of the null hypothesis.

Hypothesis XIII refers to recovery from max exercise during acute exposure to moderate altitude.

Hypothesis XIII

There is no difference between males and females in regard to altitude effect on heart rate recovery ratio.

Both sexes would be expected to require augmented recovery times, since a reduced availability of O_2 would lead to slower repayment of the O_2 debt. Females have been reported to display an accelerated loss of HCO_3^- (33) which is the primary buffer of lactic acid (LA). As a consequence, due to the resultant decrease in LA tolerance, women cannot accumulate as large an O_2 debt. Therefore, an increased recovery time was expected in both sexes at altitude but with the increase being smaller in females. Thus, rejection of the null hypothesis was anticipated.

Delimitations

1. The sample size which was statistically determined through power analysis based on Bonferroni adjusted t's consisted of 17 male and 20 female Caucasian students between the ages of 18 yrs and 24 yrs. All were non-smokers and physical education majors or minors at The University of New Mexico. A sample of this size provided an 80% probability of detecting significant differences in the variables under investigation.

2. Subjects were randomly sampled from the physical education students of record. However, they had the perogative of refusing to participate, in which case the next randomly identified student was selected.

3. All subjects were acclimatized to the altitude of 1576 m, which was the altitude selected for acquisition of base-line data.

4. The altitude increments were 1220 m from an initial altitude of 1576 m.

5. Altitudes above 1576 m were simulated in a hypobaric chamber.

6. A bicycle ergometer was used as the exercise mode and the test was of the progressive, multi-stage type, where the load was augumented every min until max effort was attained.

7. The exercise was conducted during the Spring semester at the campus of The University of New Mexico over an 11 wk period. Each subject was tested during approximately the same time of day at each altitude. The time interval between repeated tests was 2 wks.

8. Submax exercise was defined as 50% of max aerobic power at 1576 m.

9. Max effort was considered to have been reached when subjects were unable to maintain the proper pedalling rate. Further documentation of max effort was provided by an RER in excess of 1.0.

10. Males and females were not equated in terms of aerobic fitness.

Limitations

1. Subjects' daily activities outside the chamber as well as their diets were not controlled.

2. Possible variations in performance due to the menstrual cycle were not accounted for in the female subjects.

3. The climatic environment during testing could not be completely controlled. However, the temperature remained within the range of 72°-98° F and the humidity within the range of 5%-52%.

4. Testing was not conducted during the same time of day for all subjects.

5. Motivational influences exerted upon subjects by the test administrator were not completely standardized.

Definition of Terms

1. Cardiovascular recovery. Measured by the decrease in heart rate ratio for the second, fifth, and tenth min of recovery following each max exercise test.

2. Encouragement. Subjects were urged to attain maximum effort in the following manner:

a. A time goal was established for each subject, in that he was told to make every effort to continue to exercise for a given number of minutes. This number exceeded by 2 min the max exercise time attained on the base-line test.[1]

---

[1]The time goal for the first test was 18 min for women and 25 min for men. These times, while considered impossible, were chosen because they had never been reached in the Human Performance Laboratory despite attempts by world class endurance athletes. Encouragement as outlined in "b" was applied to the initial test of each subject.

b. The only verbal encouragement by the testor was to provide information regarding time remaining until the goal was reached.

3. Healthy. Determined by a recent physical examination or by a statement from a cardiologist that the resting ECG was normal.

4. Heart rate ratio. The ratio between the HR at any given time and the pre-exercise HR.

5. Maximum (max) exercise. Considered to have been attained when the pedalling rate could no longer be maintained despite encouragement and the RER exceeded 1.0.

6. Moderate altitude. Altitudes from 1500 m to 4500 m.

7. Non-smoker. A person who had not smoked more than one pack of cigarettes per month during the last 2 yrs.

8. Permanent resident. Subject whose address had been in the state of New Mexico for the last 12 mos. In addition, uninterrupted residency within the state during the preceding 6 mos was verified prior to inclusion of the subject in the study.

9. Submaximal (submax). Test data collected when $\dot{V}O_2$ was 50% of max $\dot{V}O_2$ as determined at 1576 m.

Basic Assumptions

1. Subjects in this study would be comparable in physical capacities to cadets entering the Air Force Academy.

2. Physiological responses to acute exposure to reduced barometric pressure would be similar whether the altitude is simulated or real.

3. The exercise test used was of sufficient intensity and duration to discriminate between men and women among the variables under investigation.

Need for the Study

This study was designed to clarify whether men and women would be affected similarly while exercising in an atmosphere of reduced barometric pressure. Such information is necessary to design a physical conditioning program equitable to both sexes entering the Air Force Academy or to any other group composed of both men and women undergoing aerobic training at altitude.

In addition, comparative performance evaluation of males and females at altitude will be subjective unless the knowledge which this study was designed to provide is available. Also, competition between teams consisting of both sexes is likely to become more common as emphasis on womens' athletics gains momentum. When such teams travel to higher elevations for competition, coaches will need to know whether both sexes exhibit similar responses to reduced barometric pressure. Answers to these questions would constitute an original contribution to the profession.

If men and women are indeed affected differently by altitude, new research may be generated in the area of sex differences in response to rapid decompression and variations in gravitational forces. These would be logical areas of aerospace study, since women are now being accepted for pilot training by the Navy and the Air Force.

CHAPTER II

REVIEW OF LITERATURE

Physiological changes associated with exposure to altitude represent one of the most investigated fields in environmental physiology. Much of the earlier research has dealt with effects of extreme altitudes encountered by relatively few individuals, such as mountain climbers, balloonists, and aviators flying in non-pressurized aircraft without supplemental O_2. With few exceptions (24, 39,57,64,80,81), these early works were conducted exclusively on men. When women were included as subjects, the sample size was usually quite small, making generalizations about women extremely difficult. Often the influence of sex was not investigated.

The allocation of the 1968 Olympic Games to Mexico City resulted in a worldwide interest in effects of moderate altitude on human performance and the time necessary for acclimatization. Again, the absence of female subjects in these investigations was conspicuous. This is somewhat of an anomaly in that women comprised more than 30% of the competitors in the Olympic Games at Mexico City. Thus, as in many other areas of physiology, the functional characteristics of women at altitude must be inferred rather than derived from experimental data (35).

During recent times, only two groups of investigators, Hannon *et al*. (33,34,35,36,37,38,63) and Cudkowicz *et al*. (20) focused on the effects of altitude on women. Although both groups made comparisons between men and women in functional alterations associated with altitude, only one study (20) included observations on both sexes.

A summary of changes which have been noted in men under conditions of reduced barometric pressure is presented in Figure 1. The extent to which these physiological adjustments may be generalized in women is the topic of this review.

Pulmonary Function

Shields *et al*. (63), for the purpose of assessing the acute and chronic effects of altitude on pulmonary function in females, studied eight college women at 200 m and 4300 m. Pulmonary function tests were performed initially at 200 m, and repeated after 1, 7, 30, and 65 days at 4300 m. These tests were followed by post tests two days after the return to 200 m elevation.

The reported changes in the variables studied were essentially similar, both qualitatively and quantitatively, to those commonly observed in men under similar

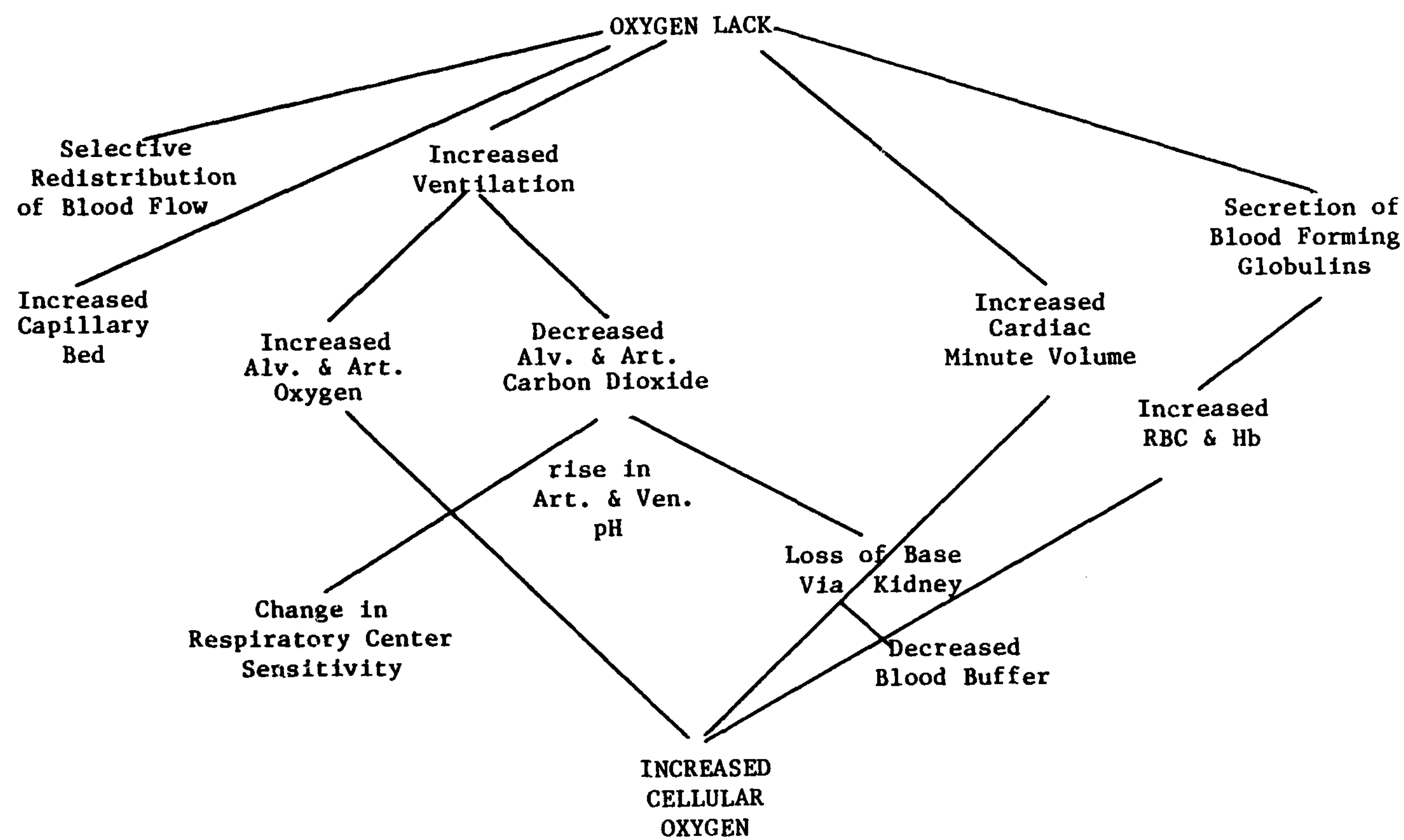

Figure 1. Overview of Physiological Effects of Environmental Hypoxia (40)

conditions. The one exception was a much smaller increase in MBC than expected from male data. After one day at 4300 m, the women's increase in MBC was 5%.

Figure 2 illustrates a comparison between the data of Shields et al. (45) and Consolazio and Issac (18), both studies having been conducted at Pikes Peak, Colorado, at 4300 m.

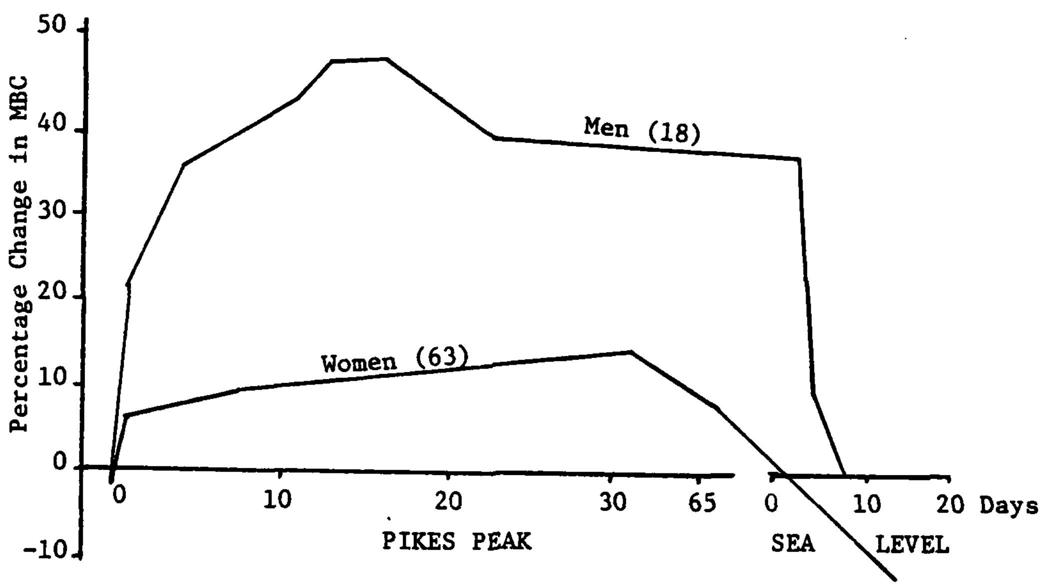

Figure 2. MBC in Men and Women at 4300 m (63)

The max increase in MBC for the men was comparable to the 40% increase found by Ulvedal et al. (70) at 4500 m. Shields et al. (45), however, found an increase of only 13.4% in women. This indicated a sex difference, since factors of age, altitude, location, equipment, testing, and correction procedures were identical. The difference between men and women in these two reports was found to be significant. Shields and co-workers stated that females might operate closer to their maximum at sea level and, therefore, have less reserve to call upon during adjustment to high altitude.

Shields _et_ _al_. (63) also found a 3.7% reduction in forced vital capacity which was significant in women only on day seven at altitude. This result was similar to decrements of 4-5% (18) and 2.4% (55) reported for men.

After the seventh day at altitude, an increase of 4% in the 1-sec forced expiratory volume (FEV_1) was also noted. Cudkowicz _et_ _al_. (20) found no difference in FEV_1 between means of 7 female and 13 male newcomers to 3600 m. However, at 5200 m, the females showed a considerable increase while the males remained virtually unchanged.

Ulvedal _et_ _al_. (70) recorded FEV_1 responses to altitude in men similar to those reported by Shields _et_ _al_. (45). The former attributed the increase in FEV_1 to reduced air density which causes a change from turbulent to laminar air flow in medium sized airways. However, since the subjects in the latter study did not have an improvment in FEV_1 until the third day, Shields _et al_. concluded that both air density and vital capacity alterations at altitude contributed to the noted improvement.

Other ventilatory differences between unacclimatized men and women residing at altitude for a period of several weeks have been reported by Cudkowicz _et al_. (20) at both 3600 m and 5200 m. Females had a larger resting $\dot{V}_A$ expressed in $l/m^2/min$, greater f, and a smaller VT expressed in ml/m^2 than did men. While the women also had higher P_AO_2, P_aO_2, and higher $\dot{V}/\dot{V}O_2$, they had lower P_ACO_2 and P_aCO_2. No difference in VDS was noted.

The foregoing research indicates that at altitude males and females would be expected to demonstrate similar responses in most pulmonary functions. An exception would be those variables dependent on or causing the exchange of large volumes of air. Thus, f and $\dot{V}$ may reach their max values earlier in females than males during activities which require max or near max effort, since women apparently operate closer to their physiological maximum for exchanging air.

Blood Composition

Changes in the blood are known to take place in a relatively short period of time on exposure to low PO_2. Gregg _et al_. (26) demonstrated that hypoxic exposure for

20 min resulted in hematocrit (Hct) elevation. Although great individual variability exists, there is roughly an increase of 10% in hemoglobin (Hb) concentration for every 100 mm drop in barometric pressure (40). The initial hemoconcentration is now generally attributed to reduction in plasma volume (35).

The same investigators (35), in an earlier unpublished study, observed Hct and Hb levels of women markedly below those generally reported in men. Since this might have been caused by inadequate iron intake, the women were given 600 mg daily supplements of $FeSO_4$ for four months prior to arrival at Pikes Peak. Upon arrival at altitude, the supplement was reduced to 300 mg per day for the remainder of the study. The dietary iron supplement was found to produce a significant increase in the Hb and Hct concentrations during the four month period prior to altitude exposure. Upon arrival at altitude, there was an immediate increase in red cell concentration. During the first 24 hrs, the Hb concentration rose from 13.6 to 14.7 g/100 ml blood. The women who had received supplementary $FeSO_4$ demonstrated Hct and Hb increases similar to those seen in men (36). A sex difference, however, may occur in the rate at which women lose, upon return to sea level, the Hb and Hct levels attained at altitude. It took the women in this study approximately 2 wks as compared to the males in another study (47) who exhibited high values for 4-5 wks after returning from 3 wks at an elevation of 4500 m.

The plasma volume demonstrated changes which were consistent with the previously noted changes in the hemoconcentration in that there was a significant decrease on the first day at altitude. The maximum change was noted after one day and was about 30% below that observed prior to altitude exposure. Hannon _et al_. (36) believed that the losses in plasma volume detected at altitude are real and not due simply to dehydration, as had been indicated by others (2,52).

During acute exposure to altitude, it is important to remember that changes in blood composition can occur within as short a period as 20 min. Unless iron supplements have been administered to women, the tendency for women to be slightly anemic should be taken into account when comparing the physiological responses of the sexes at altitude.

Serum Electrolytes

One of the first lines of defense enabling survival at altitude is the regulatory ability of the kidneys which includes: (1) increased HCO_3^- excretion, (2) decreased NH_3 formation, and (3) Cl^- conservation (40).

The electrolyte compensation for high altitude respiratory alkalosis conserves H^+, as was shown by a study in the early 1900's (32). Hurtado and Aste-Salazar (41) demonstrated that the HCO_3^- pool is soon reduced and, thus, secondary compensations must take place. This can occur either by a reduction of the concentration of one or more cations and/or by the increased concentration of one or more anions (33).

Again, Hannon _et al_. (33) provided one of the few studies in this area which used women subjects. The test location, test days, number of subjects, sex, and age group were the same as in their previously reported studies at Pikes Peak. They found two distinct phases in the acclimatization of females insofar as serum electrolytes were concerned. The first phase involved a temporary increase in the concentration of Cl^- and PO_4^{3-} which reached its maximum at 1 wk and then subsided. The second phase began while the first was still in progress and consisted of a slow reduction in Na^+ concentration and a gradual increase in the concentration of serum proteinate. The calculation of what was assumed to be a reduction of HCO_3^- showed a response in women which quantitatively was quite different from that demonstrated by men. The women in this study had a 12.1 meq/l loss of arterial HCO_3^- during the first week, as opposed to a loss of 6.0 meq/l in men under the same conditions. The loss in women, in fact, was as great in one day as that seen in men after 1 wk. This rapid loss in women indicated a greater capability for maintaining resting acid-base balance within normal range during the early stages of exposure to hypoxia. This seems to be in agreement with the finding of milder symptoms of mountain sickness in women reported by Harris _et al_. (38) and Ergueta _et al_. (23).

Comparison with a previous study by Hannon _et al_. (36) on males reveals another sex difference. In the males, the serum HCO_3^- was replaced entirely by increases in Cl^-, proteinate, and PO_4^{3-}, while in the women this loss was partially replaced through reduced Na^+ and, to a much more limited extent, K^+. Men exhibited serum osmolarities essentially unaffected by altitude, while women showed reduced serum osmolarities.

This diversity between the sexes in regulating acid-base balance might result in a difference in ventilatory responses by males and females. Other physiological functions such as O_2 debt repayment may also be affected.

Heart Rate

Concurrent with augmented ventilation, an increase in HR is the most evident response to hypoxia. Venerando _et_ _al_. (73) found an initial increase in resting HR of approximately 20% over SL values in six male athletes at 2250 m. Grollman (27), who studied the effects of altitude on one woman, found the highest resting HR on the second or third day which was approximately 30 bpm above her resting SL value.

Hannon _et_ _al_. (37,35), for the purpose of assessing the physiological effects of altitude on women during acclimatization, studied females at low elevation and then during a two month period at 4300 m. Both studies revealed sharp increases in HR of 34% and 36% on the first day at altitude. These studies showed that, after this initial increase, there was a progressive decline in HR which continued for approximately 10 wks until the pre-altitude values were reached.

Max HR shows a completely different response when compared to submax HR. Grover (28) found no reductions of consequence in HR at elevations of 3100 m. According to him, altitudes in excess of 4000 m are required for reductions in max HR. Grover and Reeves (30) reported HR values of approximately 190 ± 10 bpm in young men up to elevations of 4000 m.

The relationship between HR, SV, and $\dot{Q}$ were described by Vogel and Hansen (74). Their findings indicated that any increases in $\dot{Q}$ during rest and mild exercise resulted from increased HR. In their opinion, SV assumed a greater proportion of $\dot{Q}$ as work intensity increased. In lieu of the notion that moderate hypoxia has a direct suppressive action upon the sino auricular node or the myocardium, they proposed that, at SL, an individually optimum relationship exists between HR and SV. As altitude makes increased demands on the heart, this relationship is altered to elicit increased blood flow.

Based on the data from the foregoing studies, resting HR is expected to increase on acute exposure to reduced barometric pressure. During submax exercise, an increase in HR should occur to provide for an augmented blood flow. Max HR, however, should not be anticipated to change at altitudes of less than 4000 m.

Blood Pressure

Vogel and Hansen (74) observed the BP of 24 young males at SL and at 4300 m terrestrial altitude. They found the diastolic BP slightly, but significantly, increased at altitude. The systolic BP, although unchanged at rest, did increase during mild and moderate exercise, as well as during recovery. It dropped, however, during max work. These investigators suggested that, during hypoxic conditions, local blood flow is increased by marked vasodilation of the peripheral vascular beds. This reduces resistance to flow and maintains a relatively normal BP, while reducing the distance between the Hb molecules and active cell tissue.

Klausen _et al_. (42) also found significant, although small, increases in both systolic and diastolic BP of eight men during a 10-day sojourn at altitude (3450 m). The experimental conditions were similar to those of Hannon and Sudman (37) who investigated the basal metabolic and cardiovascular functions of females during acclimatization to altitude. In studying eight females at an elevation of 104 m followed by 80 days on Pikes Peak (4300 m), they found a significant transient increase in systolic BP and a more sustained, and also significant, increase in diastolic BP. The increase in systolic BP was immediate and amounted to approximately 15 mm Hg, while the diastolic BP was increased by 12.5 mm Hg which was reached after 36 hrs on Pikes Peak.

From these studies it appears that BP response in women is similar to that observed in men. It should be noted, however, that methodologies varied so that inter-study comparisons are of questionable validity.

Exercise Performances

No adequate studies of the exercise performance of women at altitude are available. Cudkowicz *et al.* (20), as previously discussed, did conduct a study on pulmonary function of men and women at 3600 m and 5200 m, which included exercise. There were, however, several drawbacks concerning the exercise portion. The exercise was steady-state on a bicycle ergometer and lasted only 5 min. Blood and end-tidal air were collected at the end of the fourth minute of exercise. Males and females were compared at a power load of 60 watts (367 kgm/min).

During exercise at both altitudes, the men had consistently smaller $\dot{V}DS$ and larger $\dot{V}_A$ than the women. Thus, men may have adjusted to hypoxia through a larger VT, while women responded with a higher f. The women had a smaller $\dot{V}_A/\dot{V}O_2$ at both altitudes, which suggested a greater pulmonary efficiency as compared to the men.

The women were observed to have a smaller increase in hypoxic drive, which may have been due to the minimal increase in their $PaCO_2$ during exercise. This would be the result of rising hypercapnia, especially during steady-state exercises, and could have resulted from the greater ability of the women to change VDS. This was an adaptive mechanism which differed from that found in unacclimatized males.

During an exercise period of only 5 min, very limited information on performance is attainable. In addition, the selection of an absolute, rather than relative workload for each sex makes a comparison of performance difficult, since the stronger and better conditioned subjects will be working under considerably less stress.

Dill *et al*. (21) compared three men and two women during exercise at altitude. Dill, who was 75 yrs old at the time, was one of the male subjects and another was 29 yrs old. The third male, 20 yrs of age, had undergone a pilonidal cystectomy with primary closure 2 mos prior to the study. This subject led a sedentary life for 8 wks prior to the testing. The two women were both 29 yrs of age. Due to the heterogeneity of this sample, determinations of sex differences were extremely difficult.

The effects of altitude on physical performance of women has not received much attention from physiologists. The few studies which have been conducted with female subjects have serious drawbacks which negate their generalization to larger populations.

Effects of the Menstrual Cycle

The possible interrelations between performance at altitude and the menstrual cycle were considered by

Dill _et_ _al_. (21). One of the women had a decrease in max $\dot{V}O_2$ of approximately 8% between two tests given four days apart. The second test coincided with the third day of menstruation and was also at a lower elevation than the preceding test so that the other subjects showed expected improvement. The performance of the other woman was apparently unaffected by the menstrual cycle, although at altitude her subsequent menstrual periods were shortened.

Peñaloza (50) observed that the behavior of BP at altitude in acclimatized women over 40 yrs of age was similar to that of men. However, in younger women, the systolic and diastolic BP were lower at altitude than at SL. This was thought to be due to the lesser degree of polycythemia in younger females who experienced regular blood losses. These results are not in disagreement with those reported by Hannon and Sudman (37) in that the latter provided a dietary supplement of iron to compensate for menstrual blood losses.

From a study (83) of the performance of 66 women in the Olympic Games in Tokyo and the Little Olympics in Mexico City, it was found that females won gold medals during all phases of the menstrual cycle. The premenstrum is considered by many to be the more critical period in which decrement of performance may occur (36). Sutton and Lazarus (68) reported a tendency for women to be more susceptible to mountain sickness during the premenstrual phase. This was thought to be due to a Na^+ and H_2O retention mediated by an increased secretion of aldosterone and antidiuretic hormone.

Petranyuk (51) reported that menstruation starts later in inhabitants of high altitude, beginning at 16-18 yrs of age, whereas women who migrated to altitude in early childhood began menstruation in their thirteenth to fifteenth yr. Menstrual periods were regular in both of these groups, while 27 females who had migrated to altitude after puberty suffered menorrhagia for 1-2 mos. Females whose occupation required them to ascend to altitude on a daily basis showed no change in menstrual function.

The effects of the menstrual cycle on the physical performance of females at altitude or SL appear to differ between individuals. Some women exhibit considerable decrements in performance whereas others are negligibly affected.

Summary

Studies of women indicate that sex differences in altitude response may exist. These differences indicate that women have:

A. During rest:

1. somewhat greater heart rate
2. greater respiratory frequency
3. smaller tidal volume
4. larger alveolar ventilation
5. higher ventilation equivalent for O_2
6. higher alveolar O_2 pressure
7. lower partial pressure of alveolar CO_2
8. higher arterial partial pressure of O_2
9. lower partial pressure of arterial CO_2

B. During exercise:

1. greater ventilatory dead space
2. smaller alveolar ventilation
3. smaller increase in maximum breathing capacity

C. General:

1. smaller increase in hypoxic drive
2. greater and more rapid loss of bicarbonate and a difference in its replacement.

CHAPTER III

METHODS AND PROCEDURES

Setting

Testing took place during the Spring of 1976 and was conducted in the hypobaric chamber of the Human Performance Laboratory at The University of New Mexico. This site was chosen because it allowed an exact means of controlling the barometric pressure which was an independent variable in the study.

The environmental chamber was situated at an elevation of 1576 m and is capable of simulating hyperbaric and hypobaric environments. No facilities existed for the control of temperature and humidity. Through maximum ventilation of the chamber it was possible to keep these variables within reasonable limits during testing. The chamber environment for the different test altitudes is reported in Appendix D, page 119.

Research Design

The statistical analysis procedure was an experimental design consisting of a 2 x 2 analysis of covariance (ANCOVA) with repeated measures utilizing two levels of altitude and two levels of sex. Seventeen males and 20 females each performed four exercise tests in a hypobaric chamber at a terrestrial altitude of 1576 m and simulated altitudes of 2743 m and 3962 m. A two week interval was provided between tests on the same individual. The first two tests were conducted at 1576 m to provide a reliability check for testing procedures and instrumentation. Results of this check are presented in Appendix E, page 121. The two subsequent tests were accomplished at simulated altitudes of 2743 m and 3962 m, with the order of the altitudes being determined by means of a Latin Square design. The internal validity provided by this design was considered to be critical and obtainable in that maturation, history, testing, instrumentation, statistical regression, and selection bias were adequately controlled. External validity was doubtful due to the population from which the sample was drawn. This limited generalization beyond the population used.

Source of the Data

Seventeen male and 20 female healthy college students ranging in age from 18 yrs to 24 yrs were randomly selected from majors and minors in the Department of Physical Education at The University of New Mexico. Subjects were non-smokers and premanent residents of

Albuquerque. No Negro subjects were included to avoid problems associated with sickle cell anemia. All subjects were familiarized with chamber and testing procedures through prior exposure. Selected subject data are presented in Table 1.

Method

Work tests were administered on a bicycle ergometer[1] located in the chamber. HR, BP, and f were recorded on three channels of a Physiograph Six.[2] The electrocardiogram (ECG) was recorded through two surface electrodes, BP by means of an electrosphygmograph,[2] and f was obtained from the input of a bellows pneumotach.[2]

Expired gas was collected in Douglas bags or meteorological balloons through a low resistance unidirectional Lloyd valve[3] and corrugated plastic tubing. Fractional concentrations of O_2 and CO_2 in the sampled air were determined with a Beckman E-2 fast response O_2 analyzer. With this instrument, fractional CO_2 concentrations were determined by the CO_2 scrubbing method described by Behrmann and Hartman (11). The gas analysis

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[1]Monark, Varberg, Sweden.

[2]Narco Biosystems, Houston, Texas.

[3]Collins, Braintree, Massachusetts.

TABLE 1

SELECTED SUBJECT DATA

Females				Males			
Subject	Age (yrs)	Height (cm)	Weight (kg)	Subject	Age (yrs)	Height (cm)	Weight (kg)
1	22.8	166	63	4	19.5	196	83
2	23.9	162	58	10	23.8	187	79
3	22.0	152	45	12	21.3	179	82
5	22.2	152	44	14	19.5	182	86
6	18.0	165	62	16	20.1	174	66
7	20.8	167	65	17	22.2	170	62
8	18.0	163	53	21	19.4	169	58
9	21.7	167	58	22	19.1	175	66
11	20.1	178	70	25	18.9	180	73
13	19.8	166	54	27	21.0	179	70
18	18.3	161	53	28	22.4	161	54
19	20.8	176	72	29	20.4	181	76
20	21.8	164	55	31	20.9	173	81
23	21.3	169	65	34	20.8	175	74
24	21.1	163	64	38	20.0	173	69
26	20.7	159	70	39	22.0	179	99
32	20.6	164	64	40	20.5	178	80
33	21.8	172	69				
35	19.8	169	66				
37	22.9	177	75				
$\overline{X}$	20.9	166	61	$\overline{X}$	20.7	177	74
SD	1.6	7.0	8.6	SD	1.3	7.7	11.2

was performed on flowing air samples provided by a Beckman Y-102 gas sampling unit at a constant flow rate of 70 ml/min. The instrument reading was recorded for each gas after 4 min of uninterrupted air flow. Accuracy was verified through frequent calibrations against simultaneous analyses on the Scholander Micro-Gas Analyzer. Expired air volume was determined at ATPD conditions by emptying the bag contents through a dry gas meter.[4]

Procedures

To reduce possible psychological effects of the exercise test, each subject practiced on the bicycle ergometer inside the chamber on three occasions prior to the initial testing. It has been shown (71) that, in this manner, subjects become familiar with the testing procedures and that learning reaches a plateau. On the third visit, height and gross body weight were conventionally recorded with subjects dressed in gym shorts and blouse or T-shirt. The height of the ergometer seat was individually adjusted for near maximal leg extension and the seat height recorded for use in subsequent tests. While the chamber operator depressurized[5] the chamber, each subject

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[4]American Meter Company, Philadelphia, Pennsylvania.

[5]First two tests for all subjects were conducted

remained seated on the bicycle during which time ECG electrodes, blood pressure cuff, and respiratory transducer were affixed (Appendix A, page 113). As soon as the prescribed altitude was reached, wet and dry bulb temperatures were recorded. Each subject was then told to avoid extraneous movements and to remain seated with feet positioned on the foot rests. After the subject's breathing pattern stabilized, pre-exercise data were recorded (Appendix C, page 117).

An electrical metronome was activated at 120 bpm adn the subject was instructed to pedal for approximately 20 sec to establish the pedalling rhythm. Thereafter, the exercise test (Appendix C) commenced. Cardiovascular and respiratory data were recorded during the last 15-20 sec of each min and 30-sec expired gas samples were collected periodically (Appendix C).

As HR approached 120 bpm, mouthpiece and nose clips were attached and expired gas was collected during the next minute of exercise. Thereafter, expired air was collected every other minute until three samples had been taken. Nose clips and mouthpiece were then removed until the subject showed signs of approaching max effort, i.e.,

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at ambient pressure but the vacuum pump was running so that subjects would not realize that no pressure changes were occurring.

breathing became labored and f approached pedalling rate. When this occurred, expired gas was collected during the last 30 sec of each subsequent minute. The test was terminated at the end of the minute in which the pedalling rate could no longer be maintained despite encouragement. Upon cessation of activity, the subject placed his feet on the foot rests and remained quiet for a ten minute recovery period during which HR, BP, and f were recorded every 30 sec. Toward the end of the recovery period, wet and dry bulb temperatures were again recorded.

Treatment of Data

Data from the three altitudes were treated by ANCOVA to test for differences between sexes during max and submax exercise at the two simulated altitudes. Results of the test at 1576 m served as the covariate for subsequent tests in order to control for initial sex differences for each dependent variable. To test for the simple effects of altitude between the sexes, t-tests were performed. In addition, a t-test was used to determine whether the percentage changes from one altitude to another were significantly different between the sexes.

An IBM 360 computer was used for all statistical analyses. Biomedical Computer Programs (22) and the Statistical Package for the Social Sciences (49) were used for the ANCOVA and t-tests, respectively.

CHAPTER IV

RESULTS AND ANALYSIS OF DATA

This investigation was conducted to determine differences in altitude response between males and females to selected pulmonary, cardiovascular, and metabolic variables measured during submax and max exercise. All subjects took two initial tests at 1576 m (630 mm Hg) with the one resulting in the highest max $\dot{V}O_2$ serving as the covariate for the individual's subsequent tests at 2743 m (545 mm Hg) and 3962 m (465 mm Hg). A probability level of less than 5% ($P<0.5$) was accepted as implying significance between means. Application of ANCOVA to the data provided insight as to whether differences existed between males and females in the altitude response of the variables under consideration.

The ANCOVA was used to determine the main effects and interaction effects of sex and altitude. In addition, t-tests were performed on the adjusted group means in order to test for simple effects, particularly, when altitude-sex interaction was significant. In accordance with Winer (82), the t-test for adjusted means was accomplished as follows:

$$t = \frac{\bar{X}_1 - \bar{X}_2}{\sqrt{\dfrac{\text{error } SS_1 + \text{error } SS_2}{\text{df error } SS_1 + \text{df error } SS_2}}\sqrt{\dfrac{1}{n_1} + \dfrac{1}{n_2}}}$$

The percentage change in unadjusted means between males and females from one altitude to another was also analyzed by means of a t-test.

Submaximal Work

Data referred to as submax were identified at the point when $\dot{V}O_2$ was 50% of max $\dot{V}O_2$ elicited during the base-line test at 1576 m.

Heart Rate During Submaximal Work

Table 2 shows that mean submax HR increased from 134.1 bpm to 147.5 bpm in the male group and from 141.3 bpm to 153.0 bpm in the female group. The main effect of altitude was significant. The differences of 0.6 bpm at 2743 m and 5.4 bpm at 3962 m between males and females were not significant. No significant altitude-sex interaction was detected; this verified that the HR of males and females responded similarly upon exposure to both altitudes.

Table 3 shows the total increase in submax HR to be 10.0 % in the male group and 8.3% in the female group.

TABLE 2

MEAN SUBMAXIMAL HEART RATE

Unadjusted Means and Standard Deviations

	Altitude		
	1576 m	2743 m	3962 m
Males	134.12 (12.6)	140.76 (14.8)	147.53 (16.6)
Females	141.25 (15.0)	141.40 (12.4)	152.95 (14.7)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	86.40	1	86.40	0.48	0.494
Covar	7391.33	1	7391.33	40.85	0.000
Error	6151.54	34	180.93		
Alt	1541.16	1	1541.16	38.86	0.000
Alt x Sex	105.21	1	105.21	2.65	0.112
Error	1388.00	35	39.66		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	143.61	150.38
Females	138.98	150.53
t-value[1]	1.345	0.044

[1]Critical value $t_{.05(69)} = 2.00$

There was no sex difference in these changes.

TABLE 3

PERCENTAGE CHANGE IN SUBMAXIMAL HEART RATE WITH INCREASES IN ALTITUDE

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	4.95	4.81	10.00
Females	0.11	8.17	8.28
t-value[1]	2.22	-1.56	0.43

[1]Critical value $t_{.05(37)} = 2.03$

During the first altitude increment, from 1756 m to 2743 m, the males increased by 5.0%, while the females increased by 0.1%. This constituted an altitude response which significantly differentiated between the sexes. During the second altitude increment, the males increased by 4.8% as compared to 8.2% for the females. These responses were similar. Based on these data, Hypothesis I could not be rejected.

Mean Blood Pressure During Submaximal Work

Table 4 reveals that submax $\overline{BP}$ decreased from 104.1 mm Hg to 100.4 mm Hg in the males and from 100.2 mm Hg to 94.0 mm Hg in the females when the altitude was

TABLE 4

SUBMAXIMAL MEAN BLOOD PRESSURE

Unadjusted Means and Standard Deviations

	Altitude 1576 m	2743 m	3962 m
Males	104.06 (13.9)	99.82 (12.5)	100.41 (14.3)
Females	100.20 (8.8)	94.10 (8.0)	93.95 (13.4)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	268.97	1	268.97	1.94	0.172
Covar	2996.80	1	2996.80	21.67	0.000
Error	4702.19	34	138.30		
Alt	0.88	1	0.88	0.01	0.915
Alt x Sex	2.50	1	2.50	0.03	0.857
Error	2668.33	35	76.24		

Adjusted Means

	Altitude 2743 m	3962 m
Males	98.63	99.22
Females	95.12	94.97
t-value[1]	1.031	1.247

[1]Critical value $t_{.05(69)} = 2.00$

increased from 1576 m to 3962 m. The main effect of altitude was not significant. The differences between males and females of 5.7 mm Hg at 2743 m and 6.4 mm Hg at 3962 m was insignificant. The adjusted means support the finding that the effect of altitude was similar for both sexes. There was no significant interaction between altitude and sex.

Table 5 shows the total percentage decreases in submax $\overline{BP}$ to be 3.5 mm Hg in males and 6.2 mm Hg in females. No sex differences were manifested in these changes. During the first altitude increment, from 1576 m to 2743 m, the males decreased by 4.1% as compared to 6.1%

TABLE 5

PERCENTAGE CHANGE IN SUBMAXIMAL MEAN BLOOD PRESSURE WITH INCREASES IN ALTITUDE

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	-4.07	0.59	-3.51
Females	-6.09	-0.16	-6.24
t-value[1]	0.68	-0.04	0.87

[1]Critical value $t_{.05(37)} = 2.03$

to the females. During the second altitude increment, the males increased by 0.6% as compared to a decrease of

0.2% for the females. Neither of these responses were significant. Based on these data, Hypothesis II could not be rejected.

Breathing Frequency During Submaximal Work

Table 6 shows that submax f increases from 31.8 breaths/min to 36.4 breaths/min in males and from 32.7 breaths/min to 35.5 breaths/min in females when the altitude was increased from 1576 m to 3962 m. The main effect of altitude was significant. The difference between males and females of 1.2 breaths/min at 2743 m and 0.9 breaths/min at 3962 m was not significant. The adjusted means verify that both sexes were affected similarly by altitude. There was no significant interaction between altitude and sex.

Table 7 shows the total increases in submax f to be 14.5% in the male group and 8.5% in the female group. There were no sex difference in these changes. However, during the first altitude increment from 1576 m to 2743 m the f decreased by 0.8% in females as compared to an increase of 6.0% for the males. During the second altitude increment, the females increased by 9.4% as compared to 8.0% for the males. These changes were similar in both groups. Based on these data, Hypothesis III could not be rejected.

TABLE 6

MEAN SUBMAXIMAL BREATHING FREQUENCY

Unadjusted Means and Standard Deviations

	Altitude		
	1576 m	2743 m	3962 m
Males	31.76 (4.3)	33.65 (4.5)	36.35 (6.4)
Females	32.74 (6.9)	32.47 (8.7)	35.53 (7.5)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	55.94	1	55.94	1.40	0.245
Covar	1471.45	1	1471.45	36.83	0.000
Error	1318.55	33	39.96		
Alt	148.76	1	148.76	8.95	0.005
Alt x Sex	0.54	1	0.54	0.03	0.858
Error	565.24	34	16.62		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	34.05	36.76
Females	32.11	35.16
t-value[1]	1.099	0.903

[1]Critical value $t_{.05(67)} = 2.00$

TABLE 7

PERCENTAGE CHANGE IN MEAN SUBMAXIMAL BREATHING FREQUENCY WITH INCREASES IN ALTITUDE

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	5.95	8.02	14.45
Females	-0.82	9.42	8.52
t-value[1]	0.88	-0.04	1.06

[1]Critical value $t_{05(37)} = 2.03$

Ventilation (BTPS) During Submaximal Work

Table 8 and Figure 3 show that submax $\dot{V}$ increased from 55.4 l/min to 67.1 l/min in the male group and from 37.4 l/min to 43.4 l/min in the female group when the altitude was increased from 1576 m to 3962 m. These data indicated a significant main effect of altitude. The differences between males and females of 21.2 l/min at 2743 m and 23.7 l/min at 3962 m were also significant. The adjusted means verify that the sex difference existed at both altitudes. There was no significant altitude-sex interaction.

Table 9 shows the total increases in submax $\dot{V}$ to be 21.3% in the male group and 15.9% in the female group. There was no sex difference in these changes. During the

TABLE 8

MEAN SUBMAXIMAL VENTILATION

Unadjusted Means and Standard Deviations

	Altitude		
	1576 m	2743 m	3962 m
Males	55.4 (12.2)	59.8 (12.0)	67.1 (13.2)
Females	37.4 (5.3)	38.6 (5.6)	43.4 (5.2)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	805.45	1	805.45	10.05	0.003
Covar	3085.14	1	3085.14	38.48	0.000
Error	2725.66	34	80.17		
Alt	678.59	1	678.59	60.35	0.000
Alt x Sex	29.94	1	29.94	2.66	0.112
Error	393.54	35	11.24		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	52.69	60.05
Females	44.56	49.36
t-value[1]	3.67 (P<0.001)	4.82 (P<0.001)

[1]Critical value $t_{.05(69)} = 2.00$

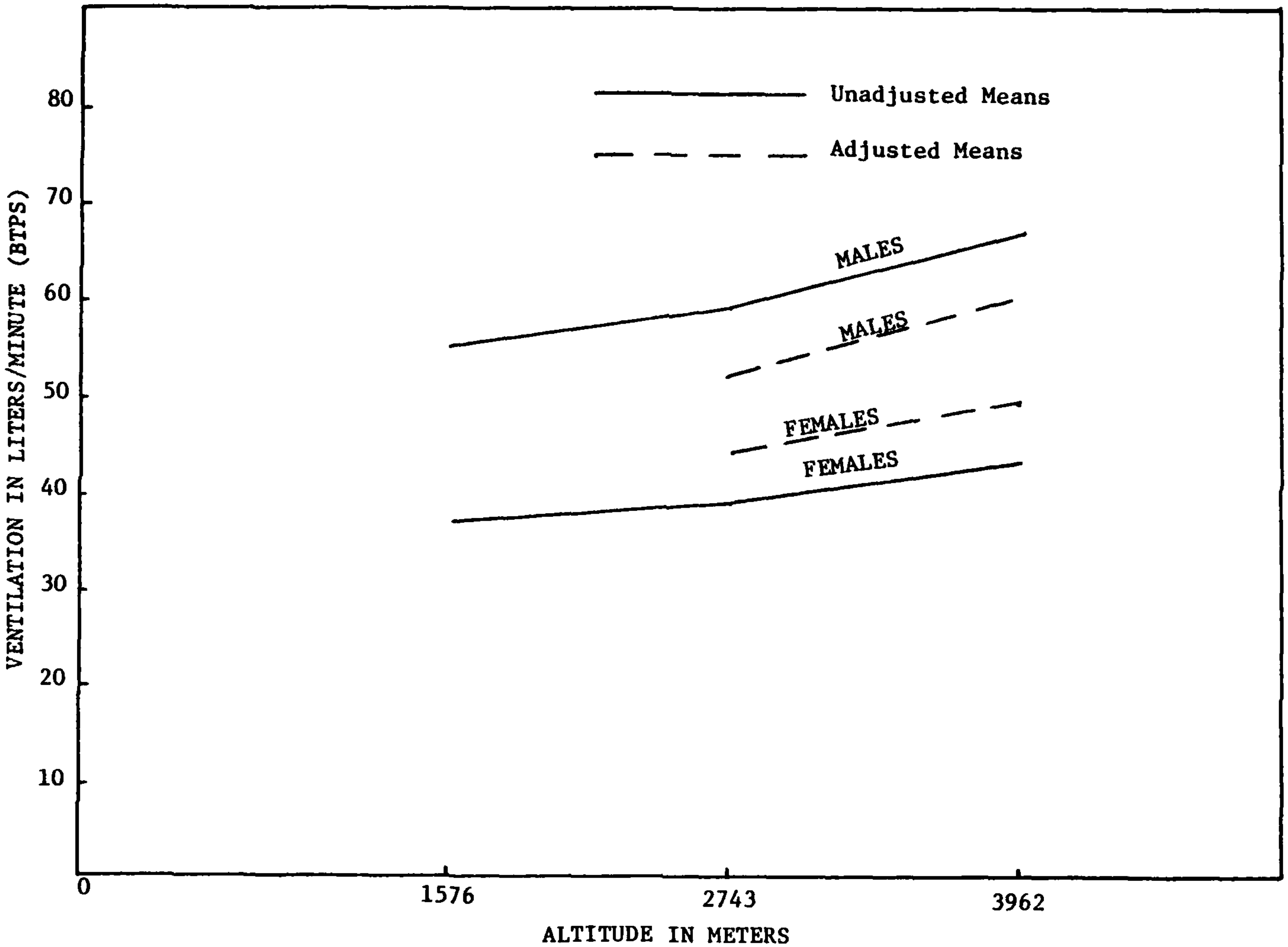


Figure 3. Ventilation During Submaximal Exercise at Three Altitudes

first altitude increment, from 1576 m to 2743 m, the males increased 8% as compared to 3.1% for the females.

TABLE 9

PERCENTAGE CHANGE IN MEAN SUBMAXIMAL VENTILATION WITH INCREASES IN ALTITUDE

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	7.97	12.32	21.26
Females	3.07	12.45	15.91
t-value[1]	1.11	-0.12	1.05

[1]Critical value $t_{.05(37)} = 2.03$

During the second altitude increment, the females increased by 12.5% as compared to 12.3% for the males. These responses were similar. Based on these data, Hypothesis IV was rejected due to difference in $\dot{V}$ between males and females which was noted in the main and simple effects of sex.

Oxygen Pulse During Submaximal Work

Table 10 shows that submax OP decreased from 19.1 ml O_2/beat to 17.5 ml O_2/beat in the male group and from 13.6 ml O_2/beat to 13.1 ml O_2/beat in the female group when the altitude was increased from 1576 m to 3962 m.

TABLE 10

MEAN SUBMAXIMAL OXYGEN PULSE[1]

Unadjusted Means and Standard Deviations

	Altitude		
	1576 m	2743 m	3962 m
Males	19.12 (2.8)	17.71 (2.4)	17.47 (2.9)
Females	13.60 (2.2)	13.85 (1.8)	13.10 (1.8)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	1.84	1	1.84	0.53	0.471
Covar	187.85	1	187.85	54.20	0.000
Error	117.85	34	3.47		
Alt	4.46	1	4.46	2.98	0.093
Alt x Sex	1.22	1	1.22	0.81	0.373
Error	52.40	35	1.50		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	15.74	15.51
Females	15.52	14.77
t-value[2]	0.425	1.428

[1]Oxygen pulse was derived from $\frac{\dot{V}O_2 \text{ in ml}}{HR} \div$ kg body wt

[2]Critical value $t_{.05(69)} = 2.00$

There was no significant main effect of altitude. The differences between males and females of 3.86 ml O_2/beat at 2743 m and 4.37 ml O_2/beat at 3962 m was not significant. The adjusted means verify that no sex differences existed at either altitude. Interaction between altitude and sex was insignificant.

Table 11 shows the decreases in submax OP to be 8.6% in the male group and 3.7% in the female group when going from 1576 m to 3962 m. There was no difference in these changes. However, during the first altitude increment, from 1576 m to 2743 m, the males <u>decreased</u> by 7.4%

TABLE 11

PERCENTAGE CHANGE IN SUBMAXIMAL OXYGEN PULSE WITH INCREASES IN ALTITUDE

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	-7.37	-1.36	-8.63
Females	1.84	-5.42	-3.68
t-value[1]	2.26	1.09	-1.46

[1]Critical value $t_{.05(37)} = 2.03$

in OP as compared to an <u>increase</u> of 1.8% for the females. This constituted a significant sex difference in altitude response. During the second altitude increment, the males decreased by 1.4% and the females by 5.4%. These differences were not significant. Based on these data, Hypothesis V could not be rejected.

Maximal Work

Oxygen Consumption (STPD) During Maximal Work

From Table 12 it can be seen that max $\dot{V}O_2$ decreased from 3.69 l/min to 3.03 l/min in the male group and from 2.44 l/min to 1.99 l/min in the female group when exercising first at 1576 m and then at 3962 m. These data indicated a significant main effect of altitude. The differences between males and females of 1.24 l/min at 2743 m and 1.04 l/min at 3962 m were not significant. The adjusted means reveal that the difference between the sexes was significant only at the lower altitudes. This resulted in the significant interaction noted in Figure 4.

Table 13 shows the total decreases in max $\dot{V}O_2$ to be approximately the same in both sexes, 17.9% and 18.4% in males and females, respectively. The percentage change at all altitudes was similar for both groups. Based on these data, Hypothesis VI was rejected because of the sex difference noted at the lower simulated altitude as well as the significant interaction.

TABLE 12

MEAN MAXIMAL OXYGEN CONSUMPTION

Unadjusted Means and Standard Deviations

	Altitude		
	1576 m	2743 m	3962 m
Males	3.69 (.47)	3.43 (.52)	3.03 (.42)
Females	2.44 (.31)	2.19 (.30)	1.99 (.31)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	0.036	1	0.036	0.56	0.460
Covar	7.854	1	7.854	122.45	0.000
Error	2.181	34	0.064		
Alt	1.665	1	1.665	84.04	0.000
Alt x Sex	0.193	1	0.193	9.75	0.004
Error	0.694	35	0.020		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	2.86	2.46
Females	2.67	2.47
t-value[1]	2.772 (P<0.01)	- .274

[1]Critical value $t_{.05(69)} = 2.00$

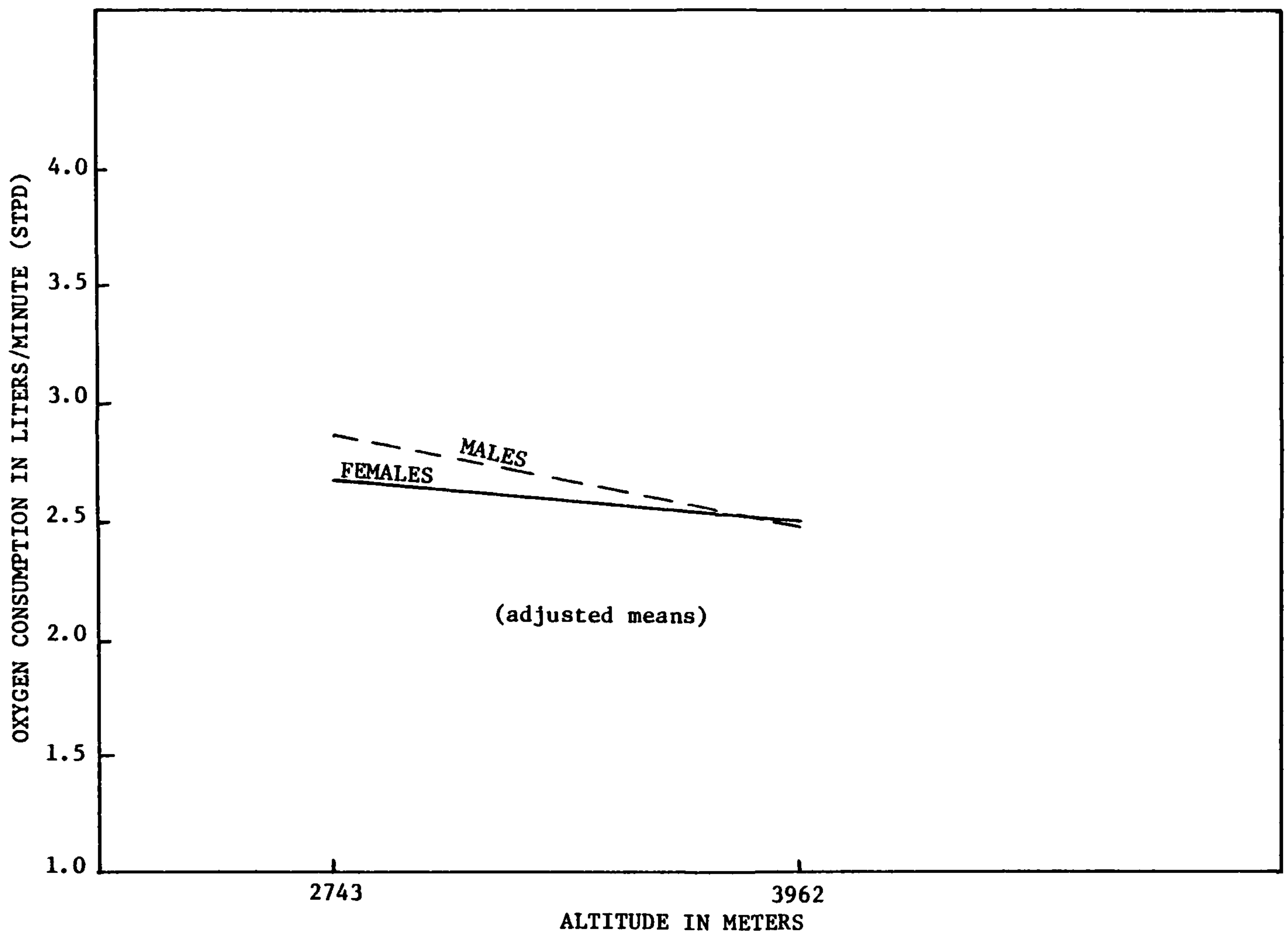


Figure 4. Altitude - Sex Interaction of Maximal Oxygen Consumption

TABLE 13

PERCENTAGE CHANGE IN MAXIMAL OXYGEN CONSUMPTION WITH INCREASES IN ALTITUDE

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	-7.05	-11.66	-17.89
Females	-10.25	- 9.13	-18.44
t-value[1]	1.78	- 1.04	0.36

[1]Critical value $t_{.05(37)} = 2.03$

Heart Rate During Maximal Work

Table 14 shows that max HR decreased from 191.8 bpm to 189.6 bpm in males and from 190.7 bpm to 186.5 bpm in females as altitude was increased from 1576 m to 3962 m. There was no significant main effect of altitude. The differences between males and females of 2.6 bpm at 2743 m and 3.1 bpm at 3962 m were not significant. The adjusted means verify the similarity between sexes in this regard. No interaction between altitude and sex was detected.

Table 15 shows the decrease in max HR to be 1.2% in males and 2.2% in females. No sex difference was apparent in these changes. During the first altitude increment, the male group decreased by 0.5 bpm and the

TABLE 14

MEAN MAXIMAL HEART RATE

Unadjusted Means and Standard Deviations

	Altitude		
	1576 m	2743 m	3962 m
Males	191.8 (7.2)	190.9 (9.7)	189.6 (7.0)
Females	190.7 (8.4)	188.3 (8.2)	186.5 (8.1)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	76.78	1	76.78	1.47	0.233
Covar	2295.85	1	2295.85	44.03	0.000
Error	1772.77	34	52.14		
Alt	45.68	1	45.68	2.07	0.159
Alt x Sex	0.92	1	0.92	0.04	0.839
Error	771.54	35	22.04		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	190.5	189.2
Females	188.7	186.9
t-value[1]	0.912	1.135

[1]Critical value $t_{.05(69)} = 2.00$

female group by 1.3 bpm. During the second altitude increment, the decreases were 0.7 bpm and 1.0 bpm for the males and females, respectively. These changes showed no sex difference. Based on these data, Hypothesis VII could not be rejected.

TABLE 15

PERCENTAGE CHANGE IN MAXIMAL HEART RATE WITH INCREASES IN ALTITUDE

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	-0.46	-0.71	-1.16
Females	-1.26	-0.96	-2.20
t-value[1]	0.53	0.21	1.09

[1]Critical value $t_{.05(37)} = 2.03$

Breathing Frequency During Maximal Work

Table 16 shows that max f increased from 53.6 breaths/min to 56.8 breaths/min in males and decreased from 52.2 breaths/min to 52.0 breaths/min in females as the altitude was increased from 1576 m to 3962 m. There was no significant main effect of altitude. The difference between the sexes of 4.8 breaths/min at both 2743 m and 3962 m also lacked significance. The adjusted means

TABLE 16

MEAN MAXIMAL BREATHING FREQUENCY

Unadjusted Means and Standard Deviations

	Altitude		
	1576 m	2743 m	3962 m
Males	53.6 (8.7)	56.9 (11.2)	56.8 (7.2)
Females	52.2 (10.7)	52.1 (10.2)	52.0 (10.3)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	280.91	1	280.91	2.95	0.096
Covar	1973.21	1	1973.21	20.73	0.000
Error	2950.91	31	95.19		
Alt	0.18	1	0.18	0.00	0.949
Alt x Sex	0.01	1	0.01	0.00	0.990
Error	1330.63	32	41.58		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	56.5	56.4
Females	52.4	52.3
t-value[1]	1.434	1.448

[1]Critical value $t_{.05(63)} = 2.00$

verify that males and females were affected similarly at both altitudes. No interaction between altitude and sex was detected.

Table 17 shows an increase in max f of 6.1% in the male group and a decrease of 0.5% in the female group. There was no significant sex difference in these changes. During the first altitude increment, from 1576 m to 2743 m, the males increased by 6.3% as compared to a decrease of 0.3% for the females. During the second altitude increment, the males and females both decreased by 0.2%. These changes were similar in both groups. Based on these data, Hypothesis VIII could not be rejected.

TABLE 17

PERCENTAGE CHANGE IN MAXIMAL BREATHING FREQUENCY WITH INCREASES IN ALTITUDE

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	6.27	-0.16	6.11
Females	-0.25	-0.23	-0.48
t-value[1]	0.80	0.12	1.47

[1]Critical value $t_{.05(37)} = 2.03$

Ventilation (BTPS) During Maximal Work

Table 17 and Figure 5 show that mean max $\dot{V}$ increased from 127.6 l/min to 135.8 l/min in the male group and from 93.4 l/min to 94.7 l/min in the female group when the altitude was increased from 1576 m to 3962 m. There was no significant main effect of altitude. The differences between the sexes of 42.6 l/min at 2743 m and 41.1 l/min at 3962 m were significant. The adjusted means verify that the sex differences did exist at both altitudes. There was no significant interaction between altitude and sex.

Table 19 shows the increases in max $\dot{V}$ to be 6.5% in the male group and 1.4% in the female group. There was no significant sex difference in these changes. However, during the first altitude increment, from 1576 m to 2743 m, the females exhibited a 3.7% <u>decrease</u> in max $\dot{V}$ as compared to a 3.9% <u>increase</u> for the males. This constituted a significant sex difference in altitude response. During the second altitude increment, the females increased by 5.3% as compared to 2.5% for the males. These responses were similar. Based on these data, Hypothesis IX was rejected due to the differences in $\dot{V}$ between the sexes which were noted in the main and simple effects as well as in the percentage change at the lower altitude increment.

TABLE 18

MEAN MAXIMAL VENTILATION (BTPS)

Unadjusted Means and Standard Deviations

	Altitude		
	1576 m	2743 m	3962 m
Males	127.6 (19.4)	132.5 (19.3)	135.8 (18.1)
Females	93.4 (11.5)	89.9 (14.8)	94.7 (19.2)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	1734.32	1	1734.32	7.20	0.011
Covar	10869.81	1	10869.81	45.12	0.000
Error	8190.31	34	240.89		
Alt	302.15	1	302.15	3.16	0.084
Alt x Sex	9.86	1	9.86	0.10	0.750
Error	3349.55	35	95.70		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	117.8	121.08
Females	102.4	107.21
t-value[1]	3.596 (P<0.001)	3.252 (P<0.01)

[1]Critical Value $t_{.05(69)} = 2.00$

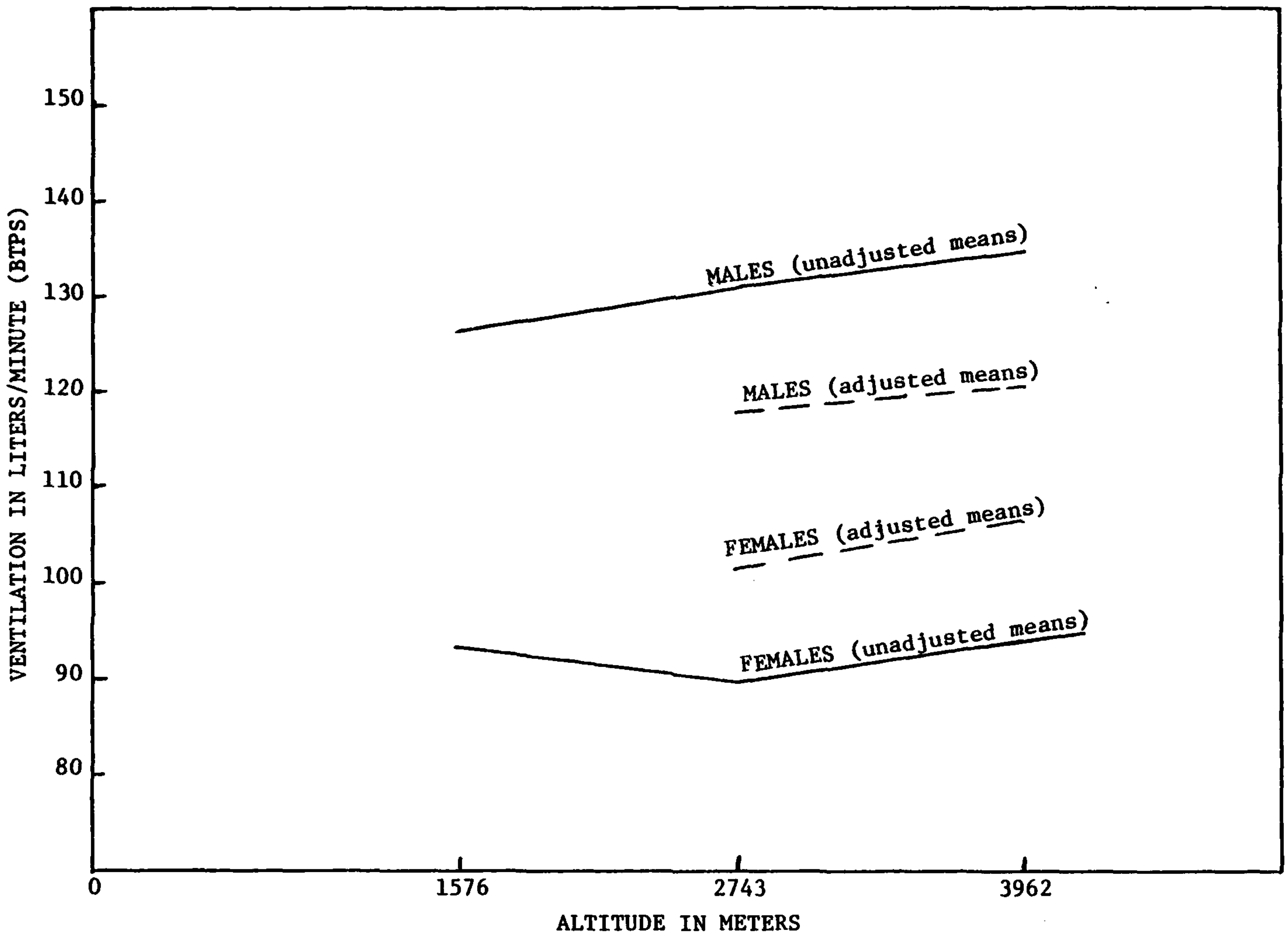


Figure 5. Ventilation During Maximal Exercise at Three Altitudes

TABLE 19

PERCENTAGE CHANGE IN MAXIMAL VENTILATION (BTPS) WITH INCREASES IN ALTITUDE

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	3.89	2.51	6.49
Females	-3.75	5.33	1.38
t-value[1]	2.24	-0.46	1.24

[1]Critical value $t_{.05(37)} = 2.03$

Oxygen Pulse During Maximal Work

Table 20 shows that max OP decreased from 26.6 ml O_2/beat to 22.4 ml O_2/beat in the males and from 21.2 ml O_2/beat to 17.4 ml O_2/beat in the females when the altitude was increased from 1576 m to 3962 m. There was a significant main effect of altitude. The differences in response between males and females of 5.84 ml O_2/beat at 2743 m and 4.91 ml O_2/beat at 3962 m were also significant. The adjusted means verify that the sex differences existed at both altitudes. There was no significant interaction between altitude and sex.

Table 21 shows the decrease in max $\dot{V}O_2$ at 3962 m to be 15.9% in the male group and 17.1% in the female group. There was no significant sex difference in this

TABLE 20

MEAN MAXIMAL OXYGEN PULSE[1]

Unadjusted Means and Standard Deviations			
	Altitude		
	1576 m	2743 m	3962 m
Males	26.58 (3.9)	25.04 (3.1)	22.35 (2.5)
Females	21.17 (2.3)	19.20 (1.8)	17.44 (1.7)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	62.83	1	62.83	16.31	0.000
Covar	198.33	1	198.33	51.48	0.000
Error	130.98	34	3.85		
Alt	91.36	1	91.36	85.80	0.000
Alt x Sex	3.97	1	3.97	3.72	0.062
Error	37.27	35	1.06		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	23.47	20.78
Females	20.54	18.77
t-value[2]	5.694 ($P<0.001$)	3.906 ($P<0.001$)

[1]Oxygen pulse was derived from $\frac{\dot{V}O_2 \text{ in ml}}{HR} \div$ kg body wt

[2]Critical value $t_{.05(69)} = 2.00$

change. During the first altitude increment, from 1576 m to 2743 m, the males exhibited a decrease of 5.8% in max OP as compared to 9.3% for the females. During the second altitude increment, the males decreased by 10.7% and the females by 9.2%. These changes were similar in both groups. Based on these data. Hypothesis XI was rejected due to the difference between males and females in OP which were noted in the main and simple effects at both simulated altitudes.

TABLE 21

PERCENTAGE CHANGE IN MAXIMAL OXYGEN PULSE WITH INCREASES IN ALTITUDE

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	-5.79	-10.74	-15.91
Females	-9.31	- 9.17	-17.13
t-value[1]	0.99	- 0.55	0.80

[1]Critical value[1] $t_{.05(37)} = 2.03$

Ventilation Equivalent for Oxygen During Maximal Work

Table 22 reveals that max $\dot{V}/\dot{V}O_2$ increased from 34.8 1/1 O_2 to 45.2 1/1 O_2 in the male group and from 38.7 1/1 O_2 to 47.6 1/1 O_2 in the female group when

TABLE 22

MEAN MAXIMAL VENTILATION EQUIVALENT FOR OXYGEN

Unadjusted Means and Standard Deviations

	Altitude		
	1576 m	2743 m	3962 m
Males	34.8 (4.6)	38.9 (4.9)	45.2 (5.4)
Females	38.7 (5.1)	41.2 (5.1)	47.6 (6.2)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	6.47	1	6.47	0.27	0.609
Covar	964.26	1	964.26	39.86	0.000
Error	822.46	34	24.19		
Alt	739.52	1	739.52	97.19	0.000
Alt x Sex	0.03	1	0.03	0.00	0.951
Error	266.32	35	7.61		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	40.5	46.82
Females	39.8	46.22
t-value[1]	0.519	0.458

[1]Critical value $t_{.05(69)} = 2.00$

exercising first at 1576 m and then at 3962 m. There was a significant main effect on altitude. The differences between males and females of 2.3 1/1 O_2 at 2743 m and 2.4 1/1 O_2 at 3962 m were not significant. The adjusted means verify that both sexes were affected similarly by each altitude used in this study. No significant interaction was detected between altitude and sex.

Table 23 shows that the increases in max $\dot{V}/\dot{V}O_2$ were 30.0% in the male group and 23.3% in the female group. There were no significant sex differences in these changes. During the first altitude increment,

TABLE 23

PERCENTAGE CHANGE IN MAXIMAL VENTILATION EQUIVALENT WITH INCREASES IN ALTITUDE

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	11.87	16.19	29.99
Females	6.51	15.48	23.00
t-value[1]	1.42	0.56	1.90

[1]Critical value $t_{.05(37)} = 2.03$

from 1576 m to 2743 m, the males increased by 11.9% in max $\dot{V}/\dot{V}O_2$ as compared to 6.5% for the females. During the second increment of altitude, the males increased by

16.2% as compared to 15.5% for the females. These changes were similar in both sexes. Based on these data, Hypothesis XII could not be rejected.

Respiratory Exchange Ratio
During Maximal Work

Table 24 reveals that max RER increased from 1.05 to 1.06 in the male group and decreased from 1.16 to 1.08 in the female group as the altitude was increased from 1576 m to 3962 m. There was no significant main effect of altitude. The differences between males and females of 0.02 at both 2743 m and 3962 m were not significant. The t-test comparing the adjusted means verify the lack of significance at both altitudes. No interaction was evident between altitude and sex.

Table 25 shows that the change in RER was increased 1.0% in the males and decreased 6.9% in the females. There was no sex difference in these changes. During the first altitude increment, from 1576 m to 3962 m, the males exhibited an increase in max RER of 1.9% as compared to a decrease of 6.0% in the females. These changes, although in different directions, were not of sufficient magnitude to show a statistically significant sex difference. During the second altitude increment, both sexes decreased 0.9%. This also lacked significance. Based on these data, Hypothesis X could not be rejected.

TABLE 24

MEAN MAXIMAL RESPIRATORY EXCHANGE RATIO

Unadjusted Means and Standard Deviations

	Altitude		
	1576 m	2743 m	3962 m
Males	1.05 (.05)	1.07 (.06)	1.06 (.08)
Females	1.16 (.15)	1.09 (.05)	1.08 (.07)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	0.012	1	0.012	1.74	0.196
Covar	0.008	1	0.008	1.14	0.293
Error	0.235	34	0.007		
Alt	0.002	1	0.002	1.07	0.308
Alt x Sex	0.000	1	0.000	0.02	0.891
Error	0.065	35	0.002		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	1.07	1.05
Females	1.09	1.08
t-value[1]	-1.265	-1.392

[1]Critical value $t_{.05(69)} = 2.00$

TABLE 25

PERCENTAGE CHANGE IN MAXIMAL RESPIRATORY EXCHANGE RATIO WITH INCREASES IN ALTITUDE

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	1.90	-0.93	0.95
Females	-6.03	-0.92	-6.90
t-value[1]	2.02	0.14	1.85

[1]Critical value $t_{.05(37)} = 2.03$

Heart Rate Recovery After Maximal Work

Recovery was defined by the HR ratio following termination of exercise as measured during the second, fifth, and tenth minutes. These data are shown in Tables 26, 27, and 28. The unadjusted mean HR ratios for males and females at 1576 m and 3962 m are displayed graphically in Figure 6. The males demonstrated a slower recovery than females at all points selected for evaluation of the recovery ratio. At these points, the main effects of altitude, sex and interaction of altitude and sex were not significant. Table 29 shows the percentage recovery and demonstrates that HR ratios drop slightly more in the men with increasing altitude as compared to the women. Based on these data, Hypothesis XIII was accepted.

TABLE 26

MEAN HEART RATE RECOVERY RATIO
SECOND MINUTE

Unadjusted Means and Standard Deviation

	Altitude		
	1576 m	2743 m	3962 m
Males	1.71 (.20)	1.63 (.21)	1.65 (.32)
Females	1.56 (.15)	1.53 (.19)	1.59 (.29)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	0.00512	1	0.00512	0.062	0.805
Covar	0.94729	1	0.94720	11.405	0.002
Error	2.82395	34	0.08306		
Alt	0.02990	1	0.02990	1.082	0.305
Alt x Sex	0.00675	1	0.00675	0.244	0.624
Error	0.96763	35	0.02765		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	1.58	1.60
Females	1.57	1.63
t-value[1]	0.012	-0.484

[1]Critical value $t_{.05(69)} = 2.00$

TABLE 27

MEAN HEART RATE RECOVERY RATIO
FIFTH MINUTE

Unadjusted Means and Standard Deviations

	Altitude		
	1576 m	2743 m	3962 m
Males	1.45 (.19)	1.40 (.19)	1.42 (.23)
Females	1.35 (.15)	1.34 (.13)	1.33 (.16)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	0.00821	1	0.00821	0.228	0.636
Covar	0.55374	1	0.55374	15.359	0.000
Error	1.22579	34	0.03605		
Alt	0.00004	1	0.00004	0.003	0.955
Alt x Sex	0.00443	1	0.00443	0.333	0.567
Error	0.46531	34	0.01329		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	1.37	1.39
Females	1.37	1.35
t-value[1]	0.127	0.728

[1]Critical value $t_{.05(68)} = 2.00$

TABLE 28

MEAN HEART RATE RECOVERY RATIO
TENTH MINUTE

Unadjusted Means and Standard Deviations

	Altitude		
	1576 m	2743 m	3962 m
Males	1.39 (.15)	1.33 (.15)	1.36 (.18)
Females	1.28 (.13)	1.25 (.16)	1.29 (.16)

ANCOVA Table

Source	SS	df	MS	F	Prob F Exceeded
Sex	0.01029	1	0.01029	0.345	0.561
Covar	0.29560	1	0.29560	9.924	0.003
Error	0.98296	33	0.02979		
Alt	0.01749	1	0.01749	1	0.308
Alt x Sex	0.00016	1	0.00016	0.010	0.922
Error	0.55475	34	0.01632		

Adjusted Means

	Altitude	
	2743 m	3962 m
Males	1.30	1.33
Females	1.28	1.31
t-value[1]	0.569	0.451

[1]Critical value $t_{.05(67)} = 2.00$

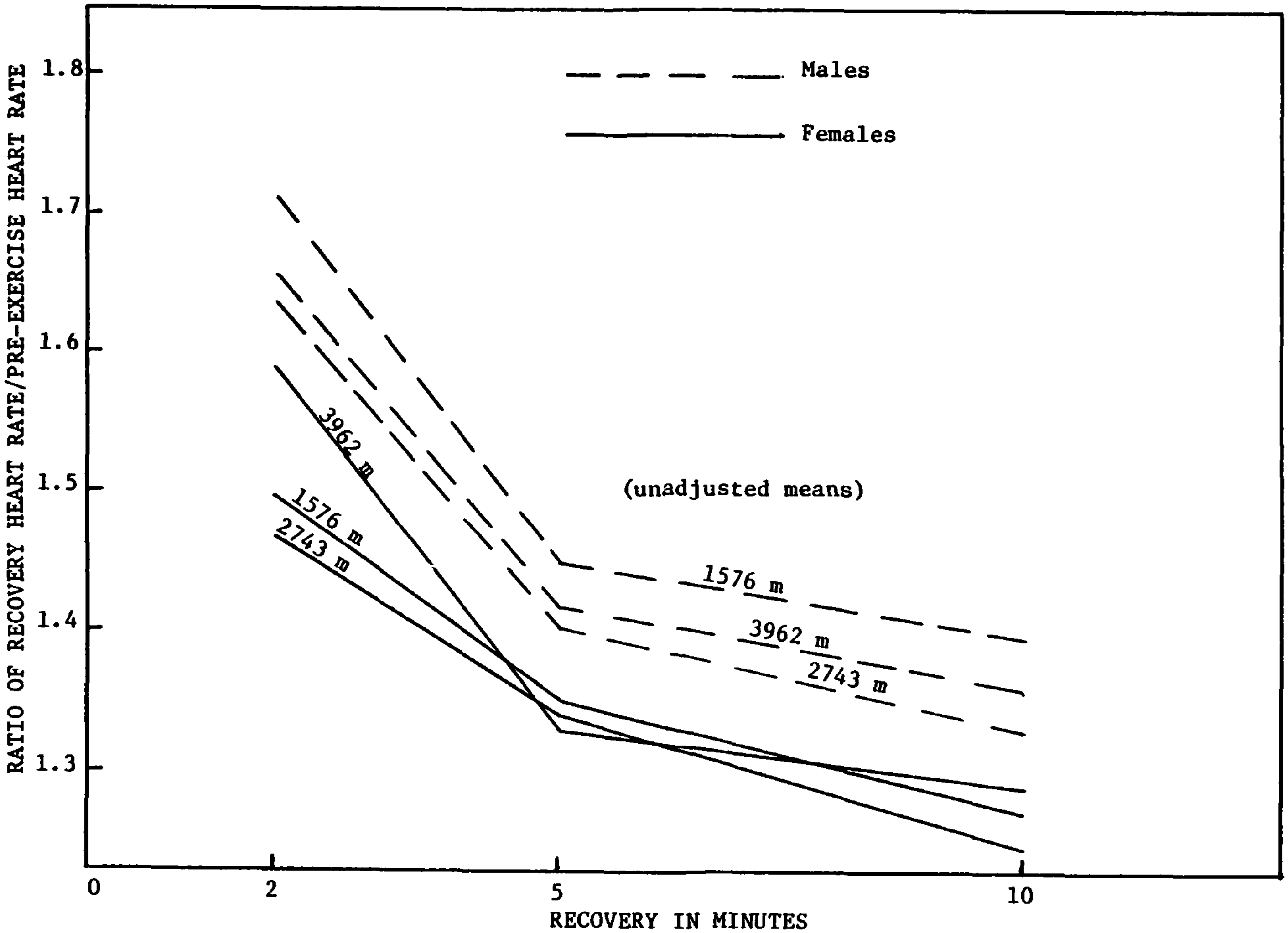


Figure 6. Heart Rate Recovery Ratio Following Maximal Exercise at Three Altitudes

TABLE 29

PERCENTAGE CHANGE IN HEART RATE RECOVERY RATIO
WITH INCREASES IN ALTITUDE

SECOND MINUTE OF RECOVERY

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	-4.68	1.23	-3.51
Females	-1.92	3.92	1.92
t-value[1]	0.10	-0.95	-1.05

FIFTH MINUTE OF RECOVERY

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	-3.45	1.43	-2.07
Females	-0.74	-0.75	-1.48
t-value[1]	0.09	0.57	-0.02

TENTH MINUTE OF RECOVERY

	Altitude Increase (meters)		
	1576-2743	2743-3962	1576-3962
Males	-4.32	2.26	-2.16
Females	-2.34	3.20	0.78
t-value[1]	-0.68	-0.21	-0.72

[1]Critical value $t_{.05(37)} = 2.03$

CHAPTER V

DISCUSSION

This study constituted an attempt to determine whether men and women would have similar responses in selected pulmonary, cardiovascular, and metabolic variables during submax and max work in an environment of reduced barometric pressure.

In an effort to minimize changes in physical fitness which could confound test results, subjects were asked to maintain their usual lifestyle for the duration of the testing period. All subjects performed max work tests on a bicycle ergometer first at 1576 m terrestrial altitude and thereafter at 2743 m and 3962 m in a hypobaric chamber. The results from the lower elevation served as covariates for adjusting data at 2743 m and 3962 m for initial differences which might have existed between the sexes. For the same reason, percentage change was also used in comparing the results from one altitude with those from another.

Submaximal Work

Data referred to as submax were collected at the point when $\dot{V}O_2$ was 50% of max $\dot{V}O_2$ as determined during the base-line test. Table 30 constitutes a summary of these data.

Heart Rate During Submaximal Work

The findings of this study concerning altitude effect on submax HR are in agreement with those of Shephard (62) on men at Mexico City. During submax work, after 2-5 days at 2300 m, the HR was slightly higher than it had previously been in Toronto (elevation 110 m). Similar HR responses have been reported by others (1,9, 17,65).

In the present study, both males and females showed increased submax HR of 10% and 8%, respectively, upon increasing the altitude from 1576 m to 3962 m. Although there was no significant sex difference in this response or in that observed between 2743 m and 3962 m, there was a significant difference between 1576 m and 2743 m. At this lower altitude increment, the females showed almost no change, while the males increased by 5%. This difference in HR response between males and females was paralleled by the changes in f and, to a lesser extent, in $\dot{V}_{BTPS}$. These data have been reproduced in Table 31 and will be discussed later.

The following is offered as a possible physiological explanation for the apparent insensitivity in the

TABLE 30

UNADJUSTED MEANS OF SELECTED VARIABLES DURING SUBMAXIMAL WORK

	1576 m		2743 m		3962 m	
	Male	Female	Male	Female	Male	Female
HR	134.1	141.3	140.8	141.4	147.5	153.0
f	31.8	32.7	33.7	32.5	36.4	35.5
$\dot{V}$	55.4	37.4	59.8	38.6	67.1	43.4
VT	1.74	1.13	1.77	1.19	1.84	1.22
$\dot{V}O_2$	1.87	1.19	1.84	1.20	1.88	1.22

TABLE 31

PERCENTAGE CHANGE IN HEART RATE, BREATHING FREQUENCY, AND VENTILATION WITH INCREASES IN ALTITUDE

	1576 — 2743 m			2743 — 3962 m		
	HR	f	$\dot{V}$	HR	f	$\dot{V}$
Males	4.95	5.95	7.97	4.81	8.02	12.32
Females	0.11	-0.82	3.07	8.17	9.42	12.45

HR response of women, when compared to men, to lower altitudes and the greater magnitude of HR response in women at higher elevations. According to Fick,

$$HR = \frac{\dot{V}O_2}{SV \times AVDO_2}$$

and, assuming a constant $\dot{V}O_2$, which was the case in this study, HR is inversely proportional to SV and $AVDO_2$. Presumably, submax HR increases at altitude due to a lower arterial O_2 saturation (SaO_2) which, in turn, results in a smaller $AVDO_2$ during exercise. HR would remain unchanged, as in the women in this study, only if either SV increased or $AVDO_2$ remained constant. SV is known to reach maximum at a HR of 110-120 bpm (5). Since in this study HR was well in excess of 120 bpm, it can therefore be assumed that SV remained unaltered. Consequently, it is likely that the women managed to maintain an adequate $AVDO_2$ despite a lower SaO_2. This could only be accomplished with a decrease in venous O_2 saturation (SvO_2).

One mechanism whereby this could be effectuated would be through an increased muscular perfusion. Women have been shown to exhibit a hyperkinetic circulation at rest and light exercise, i.e., requiring a higher $\dot{Q}$ for a given $\dot{V}O_2$ than men (5). This is caused by a lower $AVDO_2$. Provided that the necessary capillarization is available, and there is no evidence in the literature that muscles in women are less vascularized than for comparably fit men, women have a relatively greater $AVDO_2$ reserve than men.

Thus, men apparently compensated for a lower $AVDO_2$ with an increased HR, and women initially compensated for the decreased SaO_2 with an increased local perfusion resulting in a maintained $AVDO_2$ through an increased O_2 extraction. Therefore, no change in HR was required. This theory is further supported by the increased OP in females as opposed to a decrease in males at the lower altitudes. During exercise at higher altitudes, OP decreased in both sexes.

The dramatic HR increase in the females, as compared to males, at the higher altitude is explained by the possibility that $AVDO_2$, due to the aggravated hypoxia, now had normalized. The result would be an increased HR in order to compensate for the lower SaO_2. The increase would be greater in women due to their smaller SV. It may be possible that women, due to a greater reserve in $AVDO_2$ at lower elevations, are relatively less affected by lower to moderate altitudes than men. This reserve can apparently not be used during exercise at SL, since the $\dot{Q}/\dot{V}O_2$ ratio has been known to remain higher in women than men at all levels of work (5). The possibility exists, however, that the combined hypoxemia of exercise and environmental hypoxia constitute sufficient stimulus for increased local perfusion in women.

Mean Blood Pressure During Submaximal Work

The lack of altitude response in either sex was probably due to peripheral vasodilation resulting from tissue hypoxia. This reduced the peripheral resistance while compensating for the increased $\dot{Q}$. This is supported by Balke's (9) contention that there is no typical response of blood pressure to hypoxia.

Breathing Frequency During Submaximal Work

Cudkowicz _et al_. (20) reported a small decrease in f for both sexes during steady-state work when ascending from 3600 m to 5200 m. This was in contrast to the present study in which the subjects displayed increases in f. It should be noted that the altitude interval was considerably higher in the former investigation and, as mentioned, some variables are not affected below 4000 m. Although there was a main effect of altitude in the present study, there was no difference between males and females.

The question can be raised whether changes in f can be generalized to other physical activities. Astrand and Rodahl (6) have reported that bicycle riding tends to elicit an f which is fixed to the work rhythm and that, during tests with the bicycle ergometer, f often increases stepwise from 12.5 breaths/min to 50 breaths/min.

Minute Ventilation (BTPS) During Submaximal Work

The females were observed to have a lower adjusted mean $\dot{V}$ than the males at both 2743 m and 3962 m. The control mechanism for $\dot{V}$ during exercise is not known (74,79). Whereas, PaO_2, $PaCO_2$, and H^+ which stimulate $\dot{V}$ remain almost constant during exercise at SL, these variables may play a part in the regulation of $\dot{V}$ at altitude because of the reduced pressure gradients. The following is a possible explanation for the difference observed between the sexes.

At work rates not associated with lactacidemia, $\dot{V}$

exhibits a linear relationship with $\dot{V}O_2$ (76). Koyal _et al_. (44) state that a nonlinear increase in $\dot{V}$ relative to $\dot{V}O_2$ is at least, in part, due to metabolic acidosis and the related respiratory compensation. Thus, any deviation from the predicted linearity of $\dot{V}/\dot{V}O_2$ represents excess $\dot{V}$ and provides an indirect indication of metabolic acidosis.

Figure 7 illustrates that, at 50% of max $\dot{V}O_2$, the males in the present study exhibited a 90% increase in $\dot{V}$ when first exercising at 1576 m and then 3962 m. Between the same two altitudes, the females decreased by 11%. At 70% of max $\dot{V}O_2$, the excess $\dot{V}$ in the males was 73% greater at 3962 m than at 1576 m as compared to a decrease of 3% in the females. The excess $\dot{V}$ for both sexes was practically the same while exercising at 1576 m and the differences observed at higher altitudes indicated a considerably greater metabolic acidosis in the males than in the females.

At a given work load there is an earlier accumulation of lactic acid at altitude. This lowers the pH (8) which is a ventilatory stimulus (78). During exposure to altitude, women have been reported to exhibit a greater ability than men for maintaining their acid-base balance (33). This could have a damping effect on $\dot{V}$ in women.

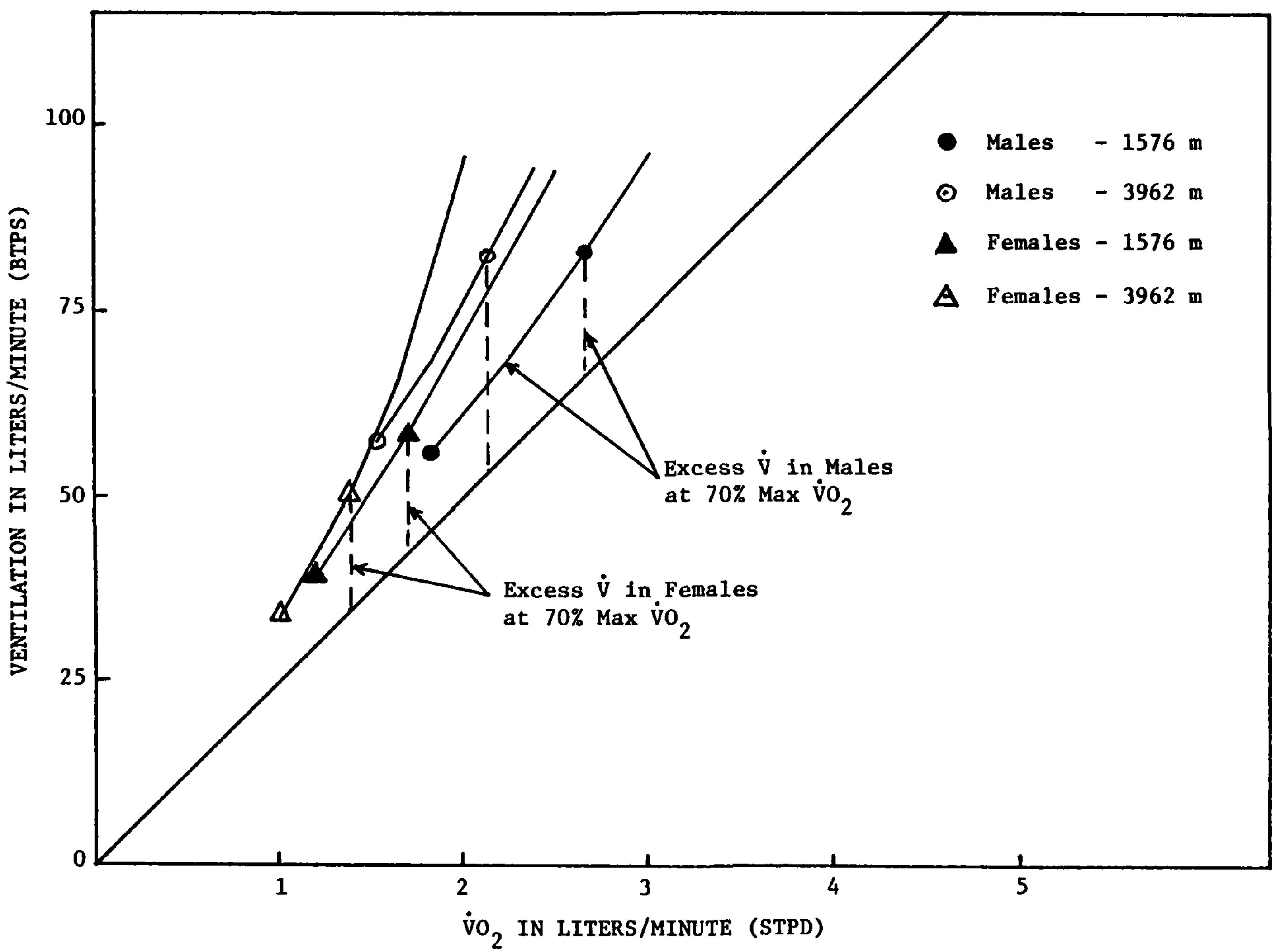


Figure 7. Excess Ventilation Stimulated by Metabolic Acidosis at 1576 m and 3962 m

Oxygen Pulse During
Submaximal Work

Submax OP was calculated from actual gas samples and, for this reason, it varied slightly from 50% of max $\dot{V}O_2$ obtained at 1576 m. Other research (17,54) indicates that $\dot{V}O_2$ for a given task is known to be independent of altitude. In the present study, submax HR increased with altitude, although no sex difference was apparent. Thus, the decrease in OP with altitude observed in this investigation was expected as well as the absence of any differences between males and females in altitude effect.

Maximal Work

Oxygen Consumption (STPD)
During Maximal Work

Table 32 provides a summary of selected variables obtained during max exercise. According to Buskirk (13), max $\dot{V}O_2$ decreases by approximately 3.2% for each 1000 ft (305 m) above 5000 ft (1525 m) elevation. Subjects in the present study would thus be expected to have a decrease of 22% in max $\dot{V}O_2$ at 3962 m, which compares well to the finding of decreases of approximately 18% in both sexes. Hagerman et al. (31) explained reductions in $\dot{V}O_2$ at 2350 m to be, in part, a result of less work of breathing due to less air density. It is likely that earlier lactacidemia and, consequently, shorter exercise time, was the primary causal agent.

TABLE 32

UNADJUSTED MEANS OF SELECTED VARIABLES DURING MAXIMAL WORK

	1576 m		2743 m		3962 m	
	Male	Female	Male	Female	Male	Female
HR	191.8	190.7	190.9	188.3	189.6	186.5
f	53.6	52.2	56.9	52.1	56.8	52.0
$\dot{V}$	127.6	93.4	132.5	89.9	135.8	94.7
VT	2.38	1.79	2.33	2.19	2.39	1.82
$\dot{V}O_2$	3.69	2.44	3.43	2.19	3.03	1.99

Although no overall sex effect in max $\dot{V}O_2$ was found in the present study, there was a difference at 2743 m which was not evident at 3962 m. These differences resulted in a significant altitude-sex interaction. This can be explained by the inability of the women to increase max $\dot{V}$ as much as the men. In addition, males and females may have been affected differently by the reduced air density. Turbulence of air flow is dependent largely upon the density, viscosity, and average velocity of the gas as well as the radius of the air passage (77). Turbulence is more apt to occur when there is a high rate of flow in a large diameter tube. The combination of larger airways and a greater relative increase in $\dot{V}$ in the men may have resulted in a more turbulent air flow while exercising at altitude. This could, in part, explain the greater decrease of max $\dot{V}O_2$ in the males between 2743 m and 3962 m. Further implications of this will be discussed in the sections for $\dot{V}$ and OP.

It has also been shown by several investigators (58,60) that individuals with high aerobic capacity have greater reductions in max $\dot{V}O_2$ at altitude. In one of these studies (60), this difference was attributed to the smaller lung diffusing capacity in athletes relative to their circulatory capacity. According to these studies, the data would indicate that the women had a greater decrease than could have been expected in men of similar fitness. Whether this represents a true sex difference on altitude response cannot be substantiated with the present data.

Heart Rate During Maximal Work

Max HR, as expected, demonstrated neither sex nor altitude effect. Max HR has been repeatedly reported to be unaffected at altitudes below 4000 m (16,30,66). Other investigators (4,53,66) have reported lower max HR at altitude in acclimatized subjects. Based on these studies, the altitudes used in the present experiment were not of sufficient magnitude to cause a reduction in either cardiac function (5) or myocardial oxygenation (12).

Breathing Frequency During Maximal Work

This variable showed no significant main effects of altitude, sex, or interaction. When using a bicycle ergometer, f often increases stepwise with increased workload. According to Astrand and Rodahl (6), for subjects within the present age range, f should be approximately 50 breaths/min during max work. In the present study, max f was 57 breaths/min for males as compared to 52 for females. From Table 32 it appears that the women reached their physiological maximum for f at 1576 m and could not increase at higher altitude despite decreased density of air. The men, however, showed a 6% increase over the 1576 m value. This could be explained by a greater capacity for ventilatory adjustment in the male subjects who were better conditioned than the females in this study. This is supported by Balke (10) who pointed out that physical work capacity at altitude is definitely restricted by the ability of the respiratory muscles to move the amount of air required.

Ventilation (BTPS) During Maximal Work

Although augmented at altitude, this variable demonstrated a blunted response during max work when compared to submax. The increases for men and women, respectively, were 6.5% and 1.4% during max work as compared to increases during submax work of 21% and 16%, respectively.

Explaining the greater increase in voluntary MBC in men as compared to women at altitude, Shields *et al*. (63) theorized that females might normally operate nearer their physiological maximum and simply have less reserve with which to make adjustments at altitude.

The present findings suggest that both sexes were able to adjust $\dot{V}$ by increasing both VT and f as compensation for the reduced PO_2 during submax work. The observations during max work may have been confounded by inequitable fitness levels due to the process by which subjects were selected for this study. Table 33 demonstrates that in this study the females were less fit than the males when classified for aerobic capacity according to Astrand (3). As shown in Table 32, page 92, the females were unable to make further adjustments in f and only slight increases in VT as air density decreased. This appears to support the findings of Shields et al. (63). Males, on the other hand, were able to increase f while VT remained practically constant. The ability of females to maintain acid-base balance, as discussed for submax $\dot{V}$, was probably also responsible for the smaller max $\dot{V}$ observed in the women. Whether these differences in max $\dot{V}$ are fitness dependent or sex dependent cannot be determined by the data generated in the present study.

Oxygen Pulse During Maximal Work

At 3962 m, males and females decreased in OP by 16% and 17%, respectively. Buskirk et al. (16) have reported a considerably larger decrease, 31% for six male collegiate trackmen when exercising at 3962 m as compared to 274 m. This disparity can probably be accounted for by the differences in fitness levels, time at altitude, and subject acclimatization.

TABLE 33

EVALUATION OF AEROBIC WORK CAPACITY BY MEANS OF OXYGEN CONSUMPTION (ML/KG/MIN)

	LOW	FAIR	AVERAGE	GOOD	HIGH
Females	$\leqq$ 28	29-34	35-43	44-48	$\geqq$ 49
Males	$\leqq$ 38	39-43	44-51	52-56	$\geqq$ 57
Females in Category	0	2 (10%)	15 (75%)	2 (10%)	1 (5%)
Males in Category	0	3 (18%)	5 (29%)	5 (29%)	4 (24%)

Since OP is a function of $\dot{V}O_2$ and HR, the aerobic fitness level of the subjects (max $\dot{V}O_2$) is reflected in max OP. Since max $\dot{V}O_2$ decreased with altitude while max HR was unaffected, the observed reduction in OP was consequential.

There was a significant difference in max OP between the males and the females in this study. Figure 8 graphically displays the adjusted means for the two variables which comprise OP. This graph demonstrates why neither $\dot{V}O_2$ nor HR had a significant main effect of sex, whereas OP did.

Ventilation Equivalent for Oxygen During Maximal Work

As seen in Figure 9, the $\dot{V}/\dot{V}O_2$ exhibited an increase in both males and females during exercise at altitude because of simultaneously increased $\dot{V}$ and decreased $\dot{V}O_2$. The only exception to this was at 2743 m when $\dot{V}$ decreased in the females. However, the ratio of decrease in the two variables was such that an increase in $\dot{V}/\dot{V}O_2$ still occurred.

The females had apparently reached their ventilatory limit at 1576 m, whereas the males demonstrated a continual increase in $\dot{V}$. The sharper drop in $\dot{V}O_2$ in males from 2743 m to 3962 m tended to reduce the differences in $\dot{V}/\dot{V}O_2$ between the sexes.

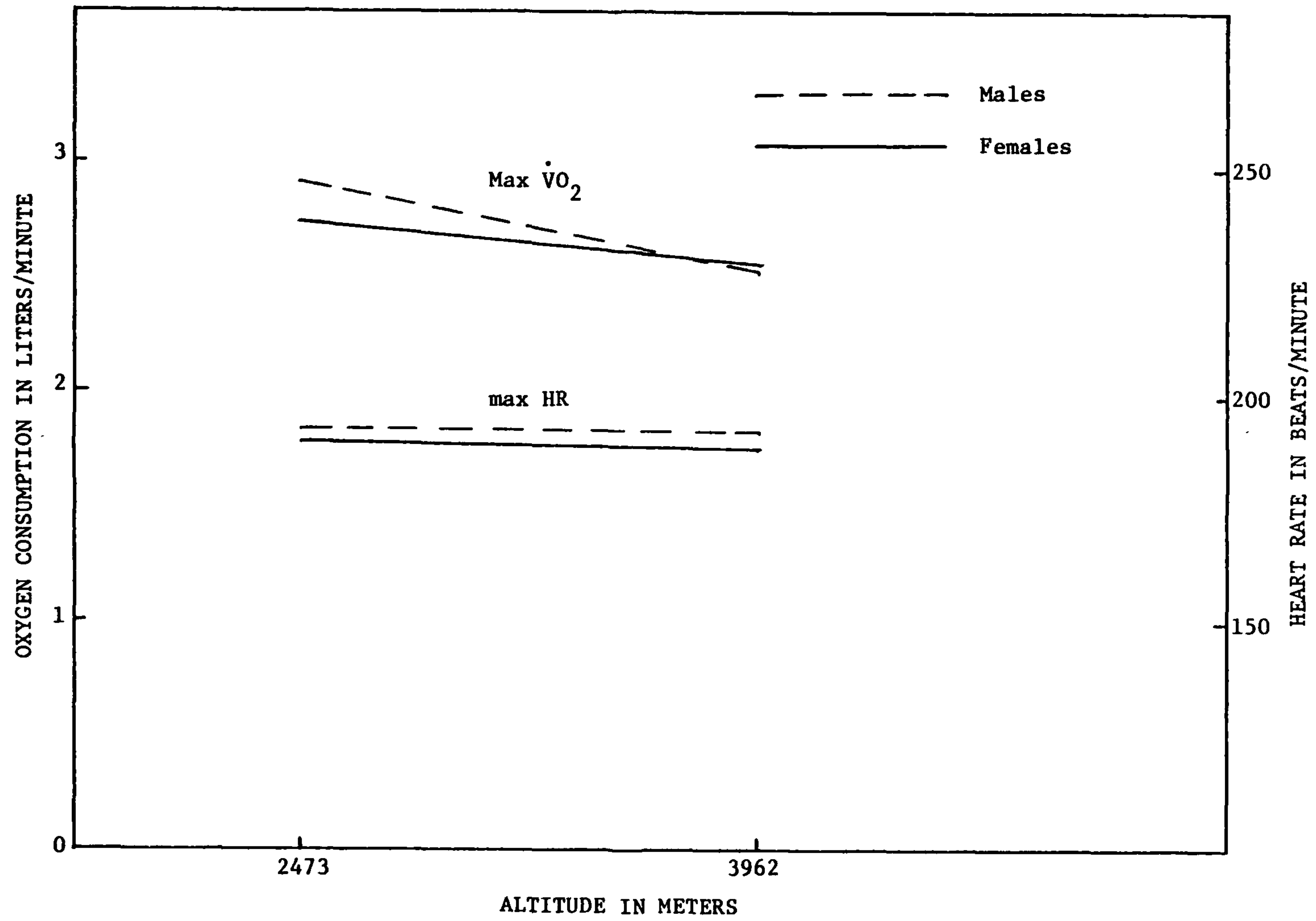


Figure 8. Adjusted Means for Maximal Oxygen Consumption and Maximal Heart Rate

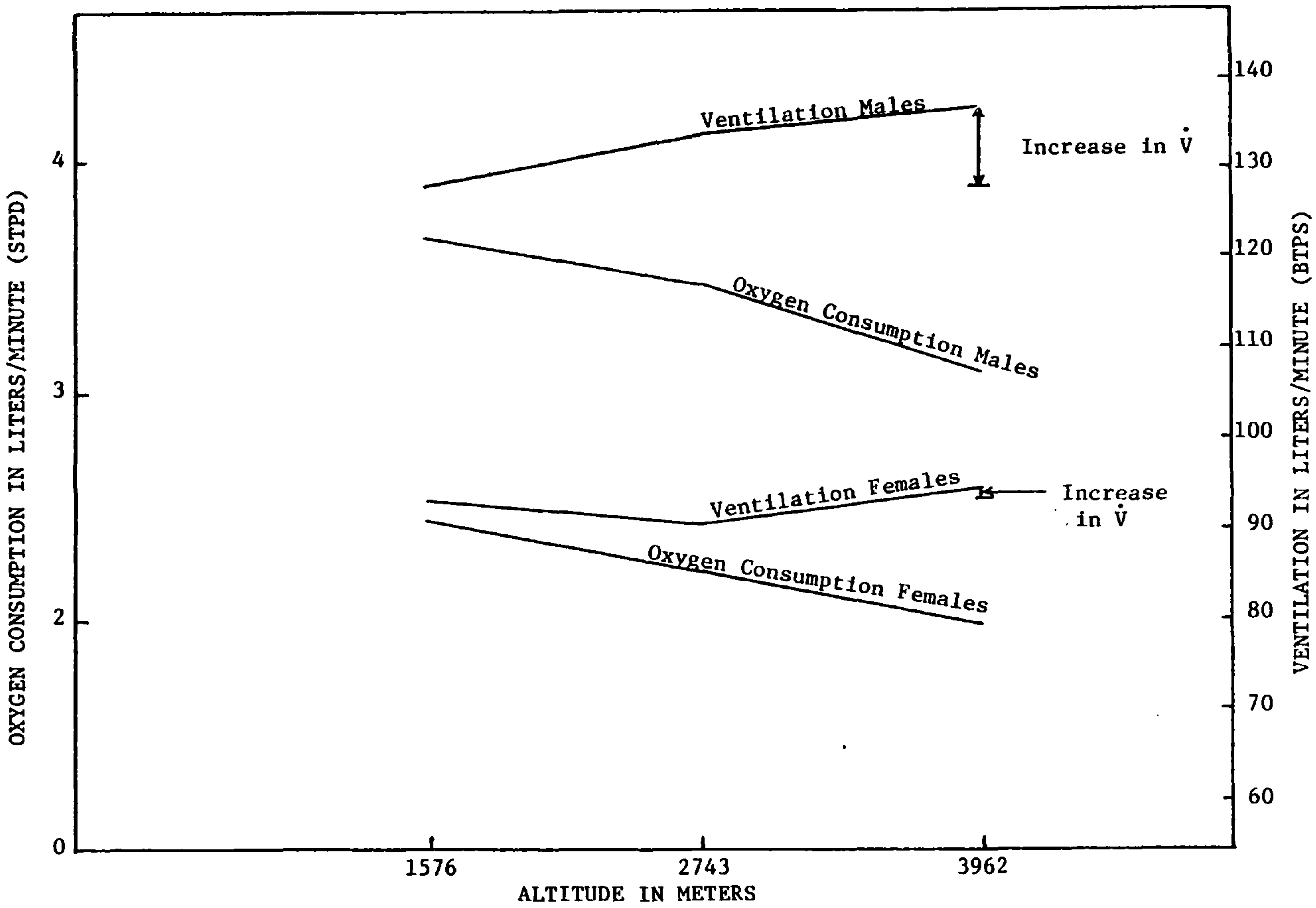


Figure 9. Unadjusted Means for Maximal Oxygen Consumption and Ventilation

The 11% higher $\dot{V}/\dot{V}O_2$ recorded for the females, as compared to the males, at 1576 m is less than half that found by McGill and Luft (46). This discrepancy was probably due to a higher aerobic capacity of the subjects in the present study resulting primarily from a smaller age span. Subjects in the former study were older and both $\dot{V}$ and $\dot{V}O_2$ are known to decrease with age (7).

Respiratory Exchange Ratio During Maximal Work

The RER exceeded 1.0 in both sexes, thus satisfying one of the criteria accepted for the study as indicating max work. At the higher altitude, the males had a small increase of approximately 1% in RER as opposed to the females who had a reduction of almost 7%. However, none of the differences, i.e., sex, altitude, interaction, or percent change, were significant.

The slightly higher RER in females observed during exposure to reduced barometric pressure was a reflection of the higher $\dot{V}/\dot{V}O_2$ observed in the women in this study. This relationship between RER and $\dot{V}/\dot{V}O_2$ has been described by Wasserman (75).

Heart Rate Recovery After Maximal Work

The increased recovery time of both sexes at altitude, which was found in the present study, has been noted by others (19,25,65). Since max O_2 debt appears to be the same at altitude as at SL (15,16) and recovery time is dependent on its repayment, the reduced inspired O_2 tension at altitude is probably responsible for the prolonged recovery.

Males demonstrated a slower recovery than females at all points selected for evaluation of the heart rate ratio, namely the second, fifth, and tenth minutes. At these points the effects of altitude, sex, and interaction were not significant.

The reason that the females showed a quicker cardiovascular recovery at altitude was probably due to their failure to accumulate as large an O_2 debt during max work. Also the lower metabolic acidosis exhibited by the women in this study apparently would support this contention.

CHAPTER VI

SUMMARY, IMPLICATIONS, AND RECOMMENDATIONS

Summary

This study was designed to investigate the effects of acute altitude exposure on selected pulmonary, cardiovascular, and metabolic variables in men and women during submax and max work.

Seventeen male and 20 female physical education students were randomly selected for inclusion in the investigation which took place over an 11-wk period. Initially, each subject took two tests at 1576 m (630 mm Hg). The test which resulted in the highest max $\dot{V}O_2$ provided data for the covariate adjustment for subsequent tests at 2743 m (545 mm Hg) and 3962 m (465 mm Hg). All testing was conducted in a hypobaric chamber with the two initial tests taking place at the ambient pressure of Albuquerque to which the subjects were already acclimated. The two subsequent tests were conducted at simulated altitudes. and the order in which these tests were given was determined by means of a Latin Square design. The following procedures were administered with a minimum of 2 wks between tests.

The subjects pedalled a bicycle ergometer at a rate of 60 rpm and a power load of 90 kpm/min for the first 3 min, after which the load was augmented by 90 kpm/min at the end of each minute until pedalling rate could no longer be maintained. When this point was reached, the test was terminated and a 10-min recovery period commenced. Heart rate (HR), blood pressure (BP), and breathing frequency (f) were recorded the last 20 sec of each minute and 30-sec samples of expired air were collected periodically until exercise was terminated. During the recovery period, HR was recorded the last 20 sec of each minute. HR was recorded during submax and max exercise as well as recovery. Minute ventilation ($\dot{V}$), f, and O_2 pulse (OP) were calculated for submax and max exercise. Mean arterial blood pressure ($\overline{BP}$) was recorded for submax exercise only. O_2 consumption ($\dot{V}O_2$), respiratory exchange ratio (RER), and ventilatory equivalent for O_2 ($\dot{V}/\dot{V}O_2$) were calculated for max exercise only.

Analysis of covariance with repeated measures was employed to test for differences between sexes during submax and max exercise at the two simulated altitudes. Results from the test at 1576 m served as the covariate for subsequent tests in order to control for initial sex differences for each dependent variable. To test for the simple effects of altitude between the sexes, t-tests were performed. In addition, a t-test was used to determine whether the percentage changes from one altitude to another were significantly different between the sexes.

Findings

Findings for submax work are summarized in Table 34 which show that the null hypotheses could not be rejected for heart rate (HR), mean blood pressure ($\overline{BP}$), ventilatory frequency (f), and oxygen pulse (OP). Rejected was the null hypothesis for minute ventilation ($\dot{V}$).

$\dot{V}_{BTPS}$ was the only variable during submax work to exhibit a consistent difference between male and female in that women had a lower $\dot{V}$ than men at both 2743 m and 3962 m. The greater ability of women to protect the acid-base balance at altitude was advanced as a possible explanation for this difference.

Neither submax HR nor OP demonstrated a difference in response due to sex from 1576 m to 3962 m. However, from 1576 m to 2743 m the percentage change of males and females in these two variables were found to be different. A theory for this apparent advantage of women as compared to men to tolerate minor hypoxic stress was advanced. It was suggested that women, at lower elevations, have latent reserve in local muscle perfusion

TABLE 34

SUMMARY OF SUBMAXIMAL RESULTS

Variable	Effects			Difference Between Adjusted Means		Difference in Percentage Change		
	Altitude	Sex	Interaction	2473 m	3962 m	1576 m - 2743 m	2743 m - 3962 m	1576 m - 3962 m
HR	yes	no	no	no	no	yes	no	no
$\overline{BP}$	no	no	no	no	no	no	no	no
f	yes	no	no	no	no	no	no	no
$\dot{V}$	yes	yes	no	yes	yes	no	no	no
OP	no	no	no	no	no	yes	no	no

which is mobilized when hypoxia reaches sufficient levels for massive vasodilation.

Table 35, which contains the findings for max work, reveals that the null hypotheses could not be rejected for HR, f, respiratory exchange ratio (RER), ventilation eqivalent for oxygen ($\dot{V}/\dot{V}O_2$), and cardiovascular recovery. Rejected were the null hypotheses for oxygen consumption ($\dot{V}O_2$), $\dot{V}$, and OP. During max work, $\dot{V}O_2$ in women was affected more by altitude than in men. This was explained by their inability to increase $\dot{V}$. Max $\dot{V}$ which remained lower in women was attributed to disparities in physiological limits, acid-base balance, O_2 cost of ventilation, and aerobic capacities between the men and women in this study. This variance in aerobic capacity as well as in airway resistance was also thought to be responsible for the aforementioned differences in $\dot{V}O_2$ and the altitude-sex interaction which was present for this variable. The difference in OP was a manifestation of the observed changes in max $\dot{V}O_2$.

Conclusions

The followng conclusions are submitted with the understanding that generalizations beyond the population sample may not be warranted:

1. During submaximal performance, environmental

TABLE 35

SUMMARY OF MAXIMAL RESULTS

Variable	Effects			Difference Between Adjusted Means		Difference in Percentage Change		
	Altitude	Sex	Interaction	2473 m	3962 m	1576 m - 2743 m	2743 m - 3962 m	1576 m - 3962 m
$\dot{V}O_2$	yes	no	yes	yes	no	no	no	no
HR	no	no	no	no	no	no	no	no
f	no	no	no	no	no	no	no	no
$\dot{V}$	no	yes	no	yes	yes	yes	no	no
OP	yes	yes	no	yes	yes	no	no	no
$\dot{V}/\dot{V}O_2$	yes	no	no	no	no	no	no	no
RER	no	no	no	no	no	no	no	no
R-2*	no	no	no	no	no	no	no	no
R-5	no	no	no	no	no	no	no	no
R-10	no	no	no	no	no	no	no	no

*second, fifth, and tenth minutes of recovery hypoxia was apparently tolerated better by women than by men.

2. During maximal performance, environmental hypoxia was apparently tolerated better by men than by women.

Implications

1. Sea level maximal aerobic performance standards should probably be reduced proportionally to a greater extent in women than in men when used at altitude testing sites.

2. Provided that exercise intensity is adjusted for individual differences in aerobic capacity, a training program of long duration and low intensity work would apparently represent a lower relative stress for women than men at altitude. Consequently, for a similar training effect in both sexes, the training intensity would probably have to be higher for women relative to their capacity than for men.

3. Women participating in aviation training programs where pressurized cockpits and supplementary oxygen are not utilized will apparently experience smaller performance decrements than men.

Recommendations

The following recommendations for further research are made:

1. The study should be repeated with males and females who are matched by physical fitness levels to determine to what extent the differences noted in this study during maximum work were fitness dependent.

2. The study should be duplicated on subjects as they acclimate to altitude to see if the differences found in this study persist.

3. In future investigations of this kind, records should be kept of the menstrual cycle to determine whether it has a confounding effect on the results.

4. The study should be duplicated with subjects from sea level in order to determine if the findings of this study are consistent over a wider range of altitudes and in people without prior acclimatization to moderate altitude.

5. Acid-base and electrolyte balance should be studied in women during exercise while chronically exposed to altitude.

6. Ventilatory response should be assessed in women during chronic exposure to altitude.

7. Active muscle arterio venous O_2 difference

should be measured in women during exercise at low-moderate altitudes.

8. Minimum training stimulus should be assessed for women, and compared to that for men, at altitude.

9. Aerobic performance norms for altitude use need to be established for both men and women relative to those currently in effect at sea level.

10. Aircrew performance in hypobaric, hypoxic environments needs to be assessed for women.

APPENDIXES

APPENDIX A

TEST INSTRUMENTATION

Test Instrumentation

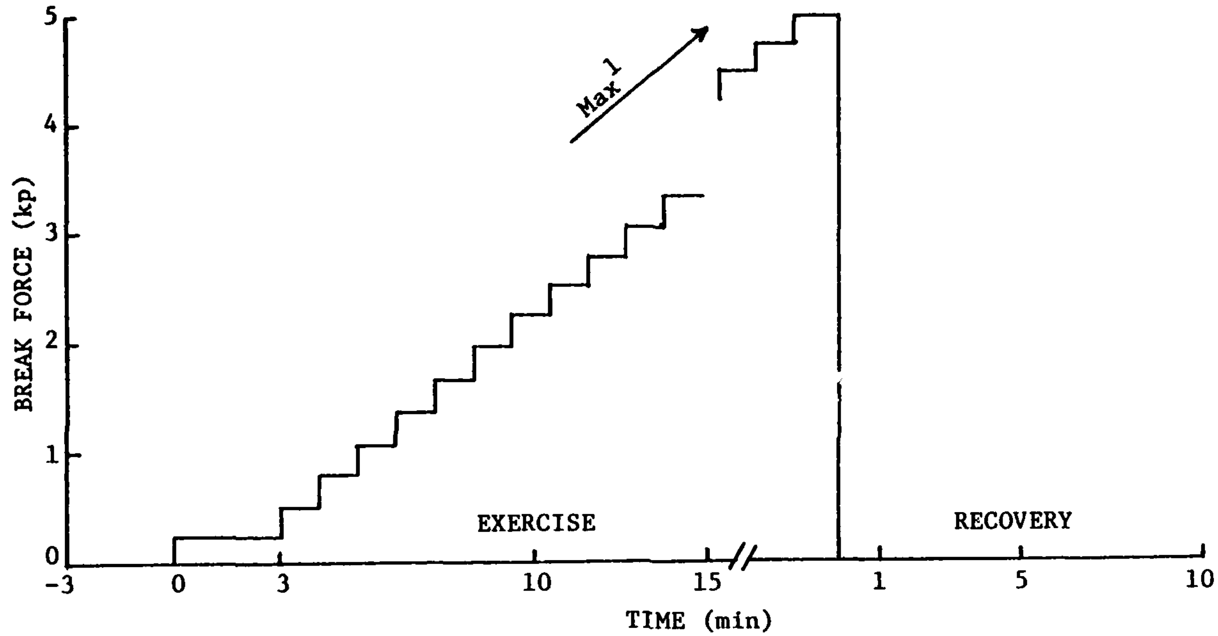
Electrocardiogram: After customary preparations, skin electrodes were affixed to the subject with the exploring electrode located above the cardiac apex in the fifth inter-costal space and the reference electrode on the manubrium of the sternum. Electrodes and input wires were further secured with adhesive tape. Paper speed was 10 mm per second.

Blood Pressure: A transducer for detecting Korotkoff sounds was attached with adhesive tape over the brachial artery on the inside of the left upper arm. A sphygmomanometer cuff was then positioned over the transducer. The cuff was inflated by a pump which was automatically activated every 60 sec. The cuff pressure reached a maximum of approximately 250 mm Hg and dropped to 20 mm Hg in about 20 sec. Prior to each test, the pressure curve was calibrated so that 1mm pen deflection equalled 5mm Hg with the Korotkoff sounds appearing superimposed upon the curve.

Ventilatory Frequency: The pneumotac (bellows-type) was strapped around the thorax.

APPENDIX B

TEST PROFILE



After the initial warm-up period of 3 min at 90 kpm/min, the power load was augmented by 90 kpm/min per min until maximum effort had been reached.

[1]The test was continued until the subject could no longer maintain the pedalling rate, at which time it was terminated and recovery commenced.

APPENDIX C

DATA COLLECTION SEQUENCE

Data Collection Sequence

Expired Air: During exercise, air samples were collected the last 30 sec of every other minute following the attainment of a heart rate in excess of 120 bpm. Three samples were collected during the submax states. As breathing became labored and ventilation frequency approached the pedalling rate, expired air samples were collected during the last 30 sec of each minute until the exercise was terminated.

Heart Rate, Blood Pressure, and Ventilation Frequency: Once the intended altitude had been reached and the subject's breathing pattern had stabilized, HR, BP, and f were recorded the last 20 sec of each minute. This procedure was continued until the termination of the 10-min recovery period.

APPENDIX D

CHAMBER ENVIRONMENT

CHAMBER ENVIRONMENT

		1576 m	2743 m	3962 m
TEMPERATURE (F)	$\bar{X}$	78.85	81.50	80.11
	SD	3.26	4.30	3.33
	Range	73-87	72-88	72-86
HUMIDITY (%)	$\bar{X}$	30.38	25.70	19.05
	SD	8.68	6.38	7.96
	Range	17-52	15-39	5-41

APPENDIX E

RELIABILITY

RELIABILITY

Submaximal Work

Variable	Means Test 1	Means Test 2	T-test 2-Tail Probability	Correlation (r)
HR	138.2	137.4	0.744	0.85
BP	98.4	100.9	0.231	0.80
f	30.7	31.4	0.232	0.96
$\dot{V}$	43.8	44.0	0.867	0.94
OP	10.3	10.7	0.184	0.97

Maximal Work

Variable	Means Test 1	Means Test 2	T-test 2-Tail Probability	Correlation (r)
$\dot{V}O_2$	2.85	2.94	0.113	0.93
HR	190.4	191.2	0.441	0.85
f	54.0	50.9	0.028	0.84
$\dot{V}$	105.1	102.0	0.173	0.94
OP	15.2	15.5	0.275	0.91

Maximal Work (continued)

Variable	Means Test 1	Test 2	T-test 2-Tail Probability	Correlation (r)
$\dot{V}/\dot{V}O_2$	29.7	34.4	0.331	0.82
RER	1.17	1.10	0.057	0.82
R-2[1]	1.62	1.60	0.517	0.80
R-5	1.40	1.40	0.902	0.77
R-10	1.33	1.34	0.763	0.74

[1]Recovery for the second, fifth, and tenth minutes

REFERENCES

LIST OF REFERENCES

1. Asmussen, E., and H. Chiodi. The effect of hypoxemia on ventilation in man. Am J. Physiol., 132:426-436, 1941.

2. Asmussen, E., and M. Nielsen. Studies on the initial increase in O_2 capacity of the blood at low O_2 pressure. Acta Physiol. Scand., 9:75-78, 1945.

3. Astrand, I. Aerobic work capacity in men and women with special reference to age. Acta Physiol. Scand., 49 (Suppl. 169):80-83, 1960.

4. Astrand, P-O., and I. Astrand. Heart rate during muscular work in man exposed to prolonged hypoxia. J. Appl. Physiol., 13:75-80, 1958.

5. Astrand, P-O., T. E. Cuddy, B. Saltin, and J. Stenberg. Cardiac output during submaximal and maximal work. J. Appl. Physiol., 19:268-274, 1964.

6. Astrand, P-O., and K. Rodahl. Textbook of Work Physiology. New York: McGraw-Hill, 1970, p. 214.

7. Ibid., p. 338.

8. Ibid., p. 566.

9. Balke, B. Variations in altitude and its effects on exercise performance. In: Exercise Physiology, edited by H. Falls. New York: Academic Press, 1968, pp. 239-265.

10. Balke, B. Work capacity and its limiting factors at high altitude. In: The Physiological Effects of High Altitude, edited by W. H. Weihe. New York: Macmillan, 1964, 233-247.

11. Behrmann, V. G., and F. W. Hartman. Rapid CO_2 determination with the Beckman O_2 analyzer. Proc. Soc. Exp. Biol. Med., 78:412-416, 1961.

12. Blomquist, G., and J. Stenberg. The ECG response to submaximal and maximal exercise during acute hypoxia. Acta Med. Scand., 178 (suppl. 440):82-93, 1965.

13. Buskirk, E. R. Decrease in physical working capacity at high altitude. In: Biomedicine Problems of High Terrestrial Elevations, edited by A. H. Hegnauer. U. S. Army Research Institute of Environmental Med., Natick, Mass., 1969, pp. 204-222.

14. Buskirk, E. R. (General discusstion.) In: Biomedicine Problems of High Terrestrial Elevations, edited by A. H. Hegnauer. U. S. Army Research Institute of Environmental Med., Natick, Mass., 1969, p. 244.

15. Buskirk, E. R., J. Kollias, R. F. Akers, B. K. Prokop, and E. Picon-Reategui. Maximal performance at altitude and on return from altitude in conditioned runners. J. Appl. Physiol., 23:259-266, 1967.

16. Buskirk, E. R., J. Kollias, E. Picon-Reategui, R. Akers, E. Prokop, and P. Baker. Physiology and performance of track athletes at various altitudes in the United States and Peru. In: The International Symposium on the Effects of Altitude on Physical Performance, edited by R. F. Goddard. Chicago: Athletic Institute, 1967, pp. 65-75.

17. Consolazio, C. F. Submaximal and maximal performance at high altitude. In: The International Symposium on the Effects of Altitude on Physical Performance, edited by R. F. Goddard. Chicago: Athletic Institute, 1967, pp. 91-96.

18. Consolazio, C. F., and G. J. Isaac. Respiratory function in normal young adults at 3475 and 4300 meters. U. S. Army Med. Res. and Nutr. Lab. Rept. No. 300, Denver, 1967.

19. Counsilman, J. E. The effects of altitude upon swimming performance. In: Proceedings of International Symposium on the Effects of Altitude on Physical Performance, edited by R. F. Goddard. Chicago: Athletic Institute, 1967, pp. 126-131.

20. Cudkowicz, L., H. Spielvogel, and G. Zubieta. Respiratory studies in women at high altitude (3,600 m or 12,200 ft and 5,200 m or 17,200 ft). Respiration, 29:393-426, 1972.

21. Dill, D. B., L. G. Myhre, D. K. Brown, K. Burrus, and G. Gehlsen. Work capacity in chronic exposures to altitude. J. Appl. Physiol., 23: 550-560, 1967.

22. Dixon, W. J. (ed.). Biomedical Computer Programs. Los Angeles: University of Calif. Press, 1974.

23. Ergueta, J., H. Spielvogel, and L. Cudkowicz. Cardio-respiratory studies in chronic mountain sickness (Monge's syndrome). Respiration, 28: 485-517, 1971.

24. FitzGerald, M. P. The changes in the breathing and the blood at various high altitudes. Phil. Trans. Roy. Soc. London, Ser. B, 203:351-371, 1913.

25. Goddard, R. F., and C. B. Favour. United States Olympic Committee swimming team performance in international sports week, Mexico City, October, 1965. In: Proceedings of International Symposium on the Effects of Altitude on Physical Performance, edited by R. F. Goddard. Chicago: Athletic Institute, 1967, pp. 135-147.

26. Gregg, H. W., B. R. Lutz, and E. C. Schneider. Changes in the content of hemoglobin and erythrocytes of the blood of man during short exposures to low oxygen. Am. J. Physiol., 50: 216-217, 1919.

27. Grollman, A. Physiological variations of the cardiac output of man. VII. The effects of high altitude on the cardiac output and its related functions: An account of experiments conducted on the summit of Pikes Peak, Colorado. Am. J. Physiol., 93:19-40, 1930.

28. Grover, R. F. (General discussion.) In: Biomedicine Problems of High Terrestrial Elevations, edited by A. H. Hegnauer. U. S. Army Research Institute of Environmental Med., Natick, Mass., 1969, p. 244.

29. Grover, R. F. Limitations of aerobic working capacity at high altitude. Adv. Cardiol., 5:11-16, 1970.

30. Grover, R. F., and J. T. Reeves. Pulmonary ventilation during exercise at altitude. In: Exercise at Altitude, edited by R. Margaria. New York: Excerpta Medica Foundation, 1967, pp. 33-39.

31. Hagerman, F. C., W. W. Addington, and E. A. Gaensler. Severe steady state exercise at sea level and altitude in Olympic oarsmen. Med. and Sci. in Sports, 7:275-279, 1975.

32. Haldane, J. S., A. M. Kellas, and E. L. Kennaway. Experiments on acclimatization to reduced atmospheric pressure. J. Physiol. (London), 53:181-206, 1919.

33. Hannon, J. P., C. W. Harris, and J. L. Shields. Alterations in the serum electrolyte levels of women during high altitude (4,300 m) acclimatization. Int. J. Biometeor., 14:201-209, 1970.

34. Hannon, J. P., J. L. Shields, and C. W. Harris. Anthropometric changes associated with altitude acclimatization in females. Am. J. Phys. Anthro., 31:77-84, 1969.

35. Hannon, J. P., J. L. Shields, and C. W. Harris. Effects of altitude acclimatization on blood composition of women. J. Appl. Physiol., 26: 540-547, 1969.

36. Hannon, J. P., J. L. Shields, and C. W. Harris. High altitude acclimatization in wome. In: Proceedings of International Symposium on the Effects of Altitude on Physical Performance, edited by R. F. Goddard. Chicago: Athletic Institute, 1967, pp. 37-44.

37. Hannon, J. P., and D. M. Sudman. Basal metabolic and cardiovascular function of women during altitude acclimatization. J. Appl. Physiol., 34:471-477, 1973.

38. Harris, C. W., J. L. Shields, and J. P. Hannon. Acute altitude sickness in females. Aerospace Med., 37:1163-1167, 1966.

39. Heber, A. R. Some effects of altitude on the human body. Lancet, 1:1148-1150, 1921.

40. Houston, C. S. Effects of high altitude (oxygen lack). In: Medical Climatology, edited by S. Licht. Baltimore: Waverly Press, 1964, pp. 469-493.

41. Hurtado, A., and H. Aste-Salazar. Arterial blood gases and acid-base balance at sea level and at high altitudes. J. Appl. Physiol., 1:304-325, 1948.

42. Klausen, K., B. Rasmussen, H. Gjellerod, H. Madsen, and E. Peterson. Circulation, metabolism and ventilation during prolonged exposure to carbon monoxide and to high altitude. Scand. J. Clin. Lab. Invest., 103:26-38, 1968.

43. Klausen, K., S. Robinson, E. D. Micahel, and L. G. Myhre. Effects of high altitude on maximum working capacity. J. Appl. Physiol., 21:1191-1194, 1966.

44. Koyal, S. N., B. J. Whipp, D. Huntsman, G. A. Bray, and K. Wasserman. Ventilatory responses to the metabolic acidosis of treadmill and cycle ergometry. J. Appl. Physiol., 40:864-967, 1976.

45. Margaria, R., and P. Cerretelli. Physiological aspects of life at extreme altitudes. In: Biometerology. Oxford, England: Pergamon Press, 1962, pp. 3-25.

46. McGill, F., and U. C. Luft. Physical performance in relation to fat free weight in women compared to men. In: Physiological Aspects of Sports and Physical Fitness. Chicago: ACSM and Athletic Institute, 1968, p. 82.

47. Merino, C. F. Studies on blood formation and destruction in the polycythemia of high altitude. Blood, 5:1-32, 1950.

48. Miller, A. T. Altitude. In: Environmental Physiology, edited by N. Balfour. St. Louis: C. V. Mosby, 1974, pp. 350-375.

49. Nie, N. H., C. H. Hull, J. G. Jenkins, K. Steinbrenner, and D. H. Bent. Statistical Package for the Social Sciences (2nd ed.) New York: McGraw-Hill, 1975.

50. Peñaloza, D. (General discussion.) In: High Altitude Physiology, edited by R. Porter and J. Knight. Edinburgh and London: Churchill Livingston, 1971, pp. 169-170.

51. Petranyuk, Y. I. Certain data from research on the sexual activity of women residents under the conditions of the high mountains. In: Problems of the Physiology and Pathology of the High Mountains. Dushanbe, Izd-vo An Tadzhikskoi SSR., 1963.

52. Pugh, L. G. C. E. Blood volume and hemoglobin concentration at altitudes above 18,000 ft (55,000 m). J. Physiol. (London), 170:344-354, 1964.

53. Pugh, L. G. C. E. Cardiac output in muscular exercise at 5800 m (19000 ft). J. Appl. Physiol., 19:441-447, 1964.

54. Pugh, L. G. C. E., M. B. Gill, S. Lahiri, J. S. Milledge, M. P. Ward, and J. B. West. Muscular exercise at great altitudes. J. Appl. Physiol., 19:431-440, 1964.

55. Rahn, H., and D. Hammond. Vital capacity at reduced barometric pressure. J. Appl. Physiol., 4:715-724, 1952.

56. Raynand, J., J. P. Martineaud, J. Bordachar, M. C. Tillous, and J. Durand. Oxygen deficit and debt in submaximal exercise at sea level and high altitude. J. Appl. Physiol., 37:43-48, 1974.

57. Reynafarje, C. The influence of high altitude on erythropoietic activity. Brookhaven Symp. Biol., 10:132-146, 1957.

58. Rimpler, A. The effect of physical fitness on the work capacity at altitude. Deutsche Forschungs- und Versuchsanstalt fur Luft-und Raumfahrt, Forschungsbericht 70-08, 1970.

59. Robinson, S. (General discussion.) In: Biomedicine Problems of High Terrestrial Elevations, edited by A. H. Hegnauer. U. S. Army Research Institute of Environmental Med., Natick, Mass., 1969, p. 244.

60. Saltin, B. Aerobic and anaerobic work capacity at an altitude of 2250 meters. In: The International Symposium on the Effects of Altitude on Physical Performance, edited by R. F. Goddard. Chicago: Athletic Institute, 1967, pp. 97-102.

61. Schilling, J. A., R. B. Harvey, E. L. Becker, T. Velasquez, G. Wells, and B. Balke. Work performance at altitude after adaptation in man and dog. J. Appl. Physiol., 8:381-387, 1956.

62. Shephard, R. J. Physical performance in Mexico City. In: The International Symposium on the Effects of Altitude on Physical Performance, edited by R. F. Goddard. Chicago: Athletic Institute, 1967, pp. 132-134.

63. Shields, J. L., J. P. Hannon, C. W. Harris, and W. S. Platner. Effects of altitude acclimatization on pulmonary function in women. J. Appl. Physiol., 25:606-609, 1968.

64. Smith, H. P., A. E. Belt, H. R. Arnold, and E. B. Carrier. Blood volume changes at high altitudes. Am. J. Physiol., 71:395-412, 1924-25.

65. Soni, J., I. C. Rivera, and F. Mendoza. The effects of altitude on non-aclimatized athletes during effort. In: The International Symposium on the Effects of Altitude on Physical Performance, edited by R. F. Goddard. Chicago: Athletic Institute, 1967, pp. 76-79.

66. Stenberg, J., B. Ekblom, and R. Messin. Hemodynamic response to work at simulated altitude, 4000 m. J. Appl. Physiol., 21:1589-1594, 1966.

67. Stiles, M. H. What medicine learned from the 1968 Olympics. Med. Times, 102:123-135, 1974.

68. Sutton, J., and L. Lazarus. Mountain sickness in the Australian alps. Med. J. Aust., 1:545-546, 1973.

69. Taggert, P., and M. Carruthers. Suppression by oxprenolol of adrenergic response to stress. Lancet, 2:256-258, 1972.

70. Ulvedal, F., T. E. Morgan, Jr., R. G. Cutler, and B. E. Welch. Ventilatory capacity during prolonged exposure to simulated altitude without hypoxia. J. Appl. Physiol., 18:904-908, 1963.

71. Unpublished data from the Human Performance Laboratory at The University of New Mexico.

72. Vanders, A. J., J. H. Sherman, and D. S. Luciano. Human Physiology: The Mechanisms of Body Functions. New York: McGraw-Hill, 1975, pp. 313-314.

73. Venerando, A., A. Dal Monte, and A. Severini. Experiences on adaptation to average altitude for subjects trained to cycle. In: The International Symposium on the Effects of Altitude on Physical Performance, edited by R. F. Goddard. Chicago: Athletic Institute, 1967, pp. 114-120.

74. Vogel, J. A., and J. E. Hansen. Cardiovascular function during exercise at high altitude. In: The International Symposium on the Effects of Altitude on Physical Performance, edited by R. F. Goddard. Chicago: Athletic Institute, 1967, pp. 47-51.

75. Wasserman, K. Testing regulation of ventilation with exercise. Chest, 70 (Suppl.):173-178, 1976.

76. Wasserman, K., A. L. Van Kessel, and G. Burton. Interaction of physiological mechanisms during exercise. J. Appl. Physiol., 22:71-85, 1967.

77. West, J. B. Respiratory Physiology: The Essentials. Baltimore: Williams and Wilkins, 1974, p. 104.

78. Ibid., p. 124.

79. Ibid., p. 127.

80. Williams, E. S. Electrolyte regulation during adaptation of humans to life at high altitude. Proc. Roy. Soc. London, Ser. B, 165:266-280, 1966.

81. Williams, E. S. Salivary electrolyte composition at high altitude. Clin. Sci., 21:37-42, 1961.

82. Winer, B. J. Statistical Principles in Experimental Design. (1st ed.) New York: McGraw-Hill, 1962, pp. 310-311.

83. Zaharieva, E. Survey of sportswomen at the Tokeyo Olympics. J. Sports Med. and Phys. Fitness, 5:215-219, 1965.

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